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TUMORS OF THE FEMALE BREAST

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(Read before the New Mexico Medical Society, at its forty-eighth Annual Meeting, Raton, N. M., June 4-6, 1930).

What is a tumor? White's descriptive definition[1]: A tumor proper is a mass of cells, tissues, or organs, resembling those normally present but arranged atypically. It grows at the expense of the organism without at the same time subserving any useful function.

Tumors of the female breast have been known for thousands of years. Cancers are mentioned in the Papyrus Ebers (1500 B.C.). Celsus, thirty years after the birth of Christ, described several varieties of cancer and he excised breast cancers, advising against the remvoal of the pectoralis major. pectoralis major.

Marcus Aurelius Severnus (1580-1650) separated cancer from benign tumors of the breast and extirpated the axillary nodes. Until the middle of the nineteenth century it was the accepted belief that all tumors of the breast were of malignant nature. When Cruveilheir, in that century, brought out the fact that many tumors of the female breast were benign in character, his statements were seriously challenged by the members of the French Academy of Medicine.

As yet, no one has ever known why tumors of any sort start in the human body or why they start in some particular spot. We accept as true the dictum that new growths never start in sound tissues. We know that chronic irritations are factors. This, however, does not explain why, in certain cases in which the source of irritation is very slight, new growths develop; while in others cases where the irritation is much more, they do not. The susceptibility of the individual here enters as a most prominent factor. In no other way may we account for the fact that 90 per cent of the human race at present do not have cancer, and that 10 per cent die from it. The studies of many investigators point to local and general susceptibility as, perhaps, the controlling factor in the genesis of malignant conditions.

"The breast is so open to observation that it is the organ from which the knowledge of the natural history of cancer was primarily derived." The mammary gland was called, by Rindfleisch, the "nurse of pathological histology."

Tumors of the breast are many and varied. We are chiefly concerned with but one classification, benign and malignant. The benign tumors are numerous and so varied as regards names that it would be but a waste of time to attempt to enumerate. Recently attempts have been made to secure a more uniform nomenclature. No one has been more active in this than McFarland. In a series of 289 cases of supposed benign fibro-epithelial tumors of the female breast he found 105 of them described under no less than 33 names. Investigations made by unbiased searchers showed them all to be periductal fibromata. One hundred and forty-seven described in much the same manner were simply mammary gland tissue, either normal or undergoing some process of involution. He concluded that all benign tumors should be classed as some form of periductal fibromata. Benign tumors of the breast give us but little concern after they are proved to be benign. The treatment of such can be given in a sentence. "Remove the segment of breast in which they are contained, but do not try to enucleate."

All suspected breast tissues should be subjected to a most rigorous examination by a competent pathologist. No one, in my opinion, has the right to pass upon the benig-

nancy of any abnormal tissue from an external examination. I know in making this statement I am to be challenged. No less an authority than Bloodgood has this to say (I quote most freely from his recent paper on "Chronic Cystic Mastitis[5]:") "The largest number of women who seek advice the moment they are warned by anything unusual in their breasts, come because of a pain, a lump, or enlargement. When carefully studied we find the largest findings are lumpy breasts which are usually due to a chronic cystic mastitis. This is the most common lesion in breast cases in which the symptoms are of one month's duration or less. In probably 70 per cent of these cases any operation can be decided against by palpation alone. In the 30 per cent in which the breast must be explored, in one half, or more, the malignancy of the lesion can be recognized by a combination of gross inspection and immediate section."

Greenough, discussing the above (and again I quote), says: "The doctrine in surgery for a long time has been that a tumor in the breast of a woman of cancerous age should be regarded as malignant until it is proved to be something else; and no matter how skillful we may think ourselves in diagnosis from an external examination, we must learn we are but human and fallible and we may be mistaken in our diagnosis."

Most cases of persistent chronic mastitis finally terminate in cancer and we are not justified in continuing the do-nothing policy in such cases. Keynes says, "Carcinoma of the breast is not necessarily preceded by chronic mastitis, but both conditions or either separately may result from one cause, namely, prolonged exposure to a chemical irritant such as may be present in the stagnating secreion in he breast."

Thus differences of opinion have existed and still will exist until the true etiology of growths is found.

The profession has a tremendous responsibility. Each one of us has to decide, not once but many times, the fate of the woman who comes to us seeking relief. The propaganda so universally adopted during the past few years has created in the mind of every intelligent female, the fear of cancer.

As previously stated, the breast is an organ open to inspection; and whether pain, a discharge of blood, or a lump, is discovered, the woman necessarily feels she must do one of two things. She either, from fear or ignorance, conceals her findings until her suffering is such that she can no longer do so, or she consults some one about her condition. For those who conceal, we can do nothing; for those who consult us, we can do a great deal. If every physician would endeavor to impress upon his female patients the very great importance of semi-annual, or even annual physical examinations, more good would be done, if accomplished, than by all the publicity in papers, public talks, and so forth. The value of the personal appeal of the family physician cannot be overestimated.

It is the speaker's opinion that we have carried "cancerphobia" too far. We have dwelt so much on the dire results to come from a lump in the breast, that many, many women put off coming to us until it is too late because they are afraid that a lump in the breast always means cancer. Our older statistics give 80 per cent of all breast tumors as being malignant. Our more recent ones show that more innocent tumors are being found than ever before. We can no longer say that the chances of a breast tumor being malignant are four to one. This fact should be stressed. We may honestly state to our patients that, if lumps are benign, there is no cause for worry if they are removed. If the tumor mass is removed and found to be malignant, the earlier a radical operation is performed the greater is the chance for cure.

During the past year, at the Pueblo Clinic we have used, in rapid diagnosis of breast tumors during operation, the method developed by Dr. Benjamin T. Terry. The growth is inspected grossly, and an appropriate portion selected and removed. This piece of tissue, about 1 by 1 by 0.5 cm. in size, is fastened by pins to a cork board, moistened with water, and cut with an ordinary razor into slices 1 mm. or less in thickness. A section is placed in a dish of distilled water and floated onto a glass slide. With the tissue surrounded by water, leaving only the upper surface free, a special neutralized polychrome methylene blue stain is applied to the upper surface with a camel's-hair brush. After a few seconds the stain is washed off with distilled water, a coverglass is applied, and the section is examined with either the 2/3 or 1/6 inch objective. The surface layer of cells is stained beautifully, while the rest of the section is translucent, and permits free passage of the light. This method is more rapid than frozen sectioning, and the tissue is more free from artefacts than when it has been frozen and thinner sections cut. The sections are not permanent, but after the emergency diagnosis has been made there is ample time for the preparation of slides from the fixed specimen.

Kocher long ago said; "The determining factor for the operative prognosis is the time and not the manner of operation."

THE MODE OF EXAMINATION

Bear in mind that tumors are likely to occur in either breast. They are more frequent in single than in married women and more common in the white than in the black race. The age of the patient must be considered. Ordinarily, after 35 is supposed to be the cancer age. However, in a series of 2083 cases reported from the Mayo Clinic,[8] 51 per cent of the cases were between the ages of 17 and 50, and 49 per cent between 50 and 84. This age incidence suggests that all tumors of the breast occurring after puberty should be regarded as possibly malignant.

In making an examination, the patient is placed flat upon her back with both hands under her head. Her chest should be absolutely bare. She should not be asked which breast is affected. The breast tissue must not be picked up, but palpated by the soft pads on the ends of the fingers, with which both breasts should be gently rolled over the underlying thorax. Any swelling, nodule, or irregularity which is discoverable clinically, will be at once felt. The object of this examination is to discover: (1) whether both breasts contain nodules, swellings, or irregularities; (2) whether the breast of which the patient complains be the only one affected, or whether there is more than one lesion.

Should multiple lesions be found in both breasts, the diagnosis is probably chronic mastitis or cystic changes, or both combnied. Should multiple swellings be found in one breast only, the diagnosis is, with some exceptions, in favor of chronic mastitis or cystic changes, and not cancer. But should only one nodule, swelling, irregularity, or even swelling surrounded by an area of irregularity, be found in one breast, it is probably a malignant tumor. Cancer of both breasts is rare, but not impossible; and a benign tumor may be found in one and cancer in the other. Hence, too much mental bias must not be allowed in any direction when only one lesion exists in each breast.

Having discovered swellings or irregularities in both breasts or only in one, the next point to be decided is, Can the skin covering the abnormal part be demonstrated to retract? This sign is best elicited by gradually and gently pushing the breast toward that part of the skin to be tested. The test is most delicate when the tumor itself is not pushed but when it is pushed indirectly by the fingers applied to a part of the breast well away from the seat of the suspected area. If present, the slightest retraction of the skin covering the suspected area of swelling can be noted. Retraction of the skin induced by this precaution is one of the earliest and most constant of the classical and clinical signs of cancer. Chealte states that, as yet, he has never seen multiple nodules, swellings or irregularities in both breasts accompanied by retraction of the covering skin.

This retraction of the skin is caused by the invasion by the cancer of the fibrous bands which pass from the breast capsule to the skin. Later it may be caused by the adherence of the cancer to the skin itself or it may be caused by the attachment of the tumor to the underlying fascia. Retraction of the nipple as a sign is too much sought for, too long waited for, and, when found, is over estimated. It occurs in cancer early when the disease begins under or near the nipple. Otherwise it is comparatively late.

Hemorrhage or discharge from the nipple must always be regarded as a dangerous sign when no inflammation or obvious cause is present to account for it.

Nearly all papers dealing with tumors of the breast stress the importance of early diagnosis. Teachers of surgery and authors of text-books must inform students and doctors how a diagnosis of early malignancy is made, if it can be done. If we wait for deformity, retraction of the nipple, pig-skin dimpling, and axillary gland enlargement, we have waited too long. Alleviation at this time is probable, cure is impossible.

It is an axiomatic fact in the light of our present knowledge, that a benign growth demands excision and a malignant one the most radical operation the surgeon is capable of performing.

In a statistical study of cancer of the breast made by H. B. Wood,[9] of Harrisburg, Pa. we obtain the pertinent information that cancer is the fourth greatest cause of death in the state; and that, between the ages of 40 and 60, it comes next to heart diseases in the destruction of life. Malignant tumors were credited with causing 8.3 per cent of all the deaths in 1927, and tumors of the breast caused 8.8 per cent of the deaths from all malignant tumors. In Pueblo, for the years 1927, 1928, and 1929, there were a total of 1723 deaths from all causes. Of this number there were 115 deaths from malignant tumors, 8 per cent of which were tumors of the breast. Deaths from cancer of the breast are increasing 3 per cent annually.

A surprising fact is the great time between the discovery of the growth and the advent for operation. Two hundred and twenty-three cases analyzed by J. J. Buchanan of Pittsburgh, gave an average of one year, five months and five days from the time growth was known to have existed.

The average life period for a cancer of the breast is about three years. The average duration of life after operation is greater by two years than that of unoperated cases from onset to death.

Metastasis may occur anywhere. Operations must be done before it begins. Harrington[7] in 1858 cases states: "The best surgical results are obtained from primary radical operations—in cases without lymphatic involvement." In this group, 80.23 per cent are living after three years; 67.44 per cent after five years; and 52.94 per cent after ten years.

About half now going to the hospital are seemingly cured. These results are probably more favorable than in malignant lesions elsewhere in the body, with the possible exception of basal cell epithelioma of the face and lips, mixed tumors of the salivary glands, and encysted papillary malignant growths of the ovary. Harrington does not believe these results will be greatly increased by more extensive operation than the radical ones now generally employed. The results where lymphatic metastases exist at the time of operation are not so good. Of this group, 41.70 per cent lived three years; 25.75 per cent five years; and 12.23 per cent ten years. He states that it is unfortunate that this latter group comprised 64.37 per cent of all operated upon, and there has been little improvement in this percentage in the last ten years. In fact, it has gradually increased. Of this series of 1859 cases, 953 patients are known to have died from metastasis; 381 had local and distant metastasis, and 572 had distant matastasis but did not have local recurrence in skin or axilla. This again emphasizes the importance of early diagnosis and operation. In order of frequency metastasis occurs in: (1) supraclavicular region; (2) lungs, mediastinum and sternum; (3) abdomen; (4) spine, femur, pelvic bones and skull; (5) opposite breast and axilla. Whenever a patient who has had cancer of the breast begins to complain of backache, beware. In the great majority of cases spinal involvement has begun.

TREATMENT

Time will not permit any discussion of operative technic, the history of its evolution, or the opinions of operators concerning relative merits. We shall pass rapidly and briefly to other modes of treatment now in use. Burton J. Lee,[8] N. Y. Memorial Hospital, says, "Five-year results with irradiation alone or combined with radical operations, gives a higher percentage of five-year results than radical surgery alone. High voltage should be employed three or four weeks before operation and again post-operatively."

Treatment of primary inoperable cancer of the breast by radiation gives relief from pain, heals superficial ulcers, improves general condition, and prolongs life. We have found this to be true in cases of this type treated at our Clinic.

Portman,[9] of the Cleveland Clinic, says intensive cross-fire irradiation is harmful and will increase the early recurrences; but, by the employment of repeated doses of moderate postoperative radiation, the number of three-year survivals will be increased.

L. B. Morrison[10] says that inoperable cancers of the breast live at least one and one half years longer following roentgen therapy and are much more comfortable. He also believes that preoperative radiation, if properly used, increases the chances of a surgical cure.

On the opposite side of the question as to the value of x-ray, we quote: "Preoperative x-ray treatment has little to recommend it. Experiments in animals have shown that the lymphatics cannot, but the terminal arteries can, be closed by its use. Therefore, to use x-ray preliminary to operation on the assumption that it will make operation safer by closing the lymphatics, is wrong if we believe in the spread of cancer by the permeation method. On the other hand, if we believe in the embolic theory, it may do good." (Wood[6]).

Infection following operation is said to occur more frequently when x-ray is used preoperatively.

Harrington[7] informs us that in his series of 1859 cases, the average length of life of all with metastasis treated by x-ray was twenty-seven months. The average of a similar group not receiving x-ray was thirty-eight months. These results show the ineffectiveness of the x-ray in metastasis or recurrent lesions, and again emphasize the importance of complete removal at the operation.

In the January, 1930, issue of the American Journal of Roentgenology, there is an abstract by Herman Wentz on "Results of Radiation Therapy in Uterine and Mammary Carcinoma." The method employed by this author is that of roentgen therapy alone. He divides his breast cases, totaling 625, into three classes:

1. Operable: 72 cases, 79 per cent, symptom-free at the end of three years; 66 cases, 53 per cent, symptom free at the end of five years.

2. Inoperable mammary carcinoma: 65 cases, 20 per cent, symptom-free at the end

of three years; 50 cases, 12 per cent symptom-free at the end of five years.

3. Irradiation in recurrences: 165 cases, 34 per cent, symptom-free at the end of five years; 136 cases 19 per cent, symptom-free at the end of five years; 71 cases, 10 per cent, symptom-free at the end of eight years.

He states with positiveness that the more advanced cases yield better results when treated by x-ray than by surgery alone.

Our foreign brothers appear to favor the use of x-ray and radium in the treatment of breast cancers.

Just what the end results will eventually be, no one can forecast; but that a cure will be found, is the earnest prayer of all.

CONCLUSIONS

1. All practitioners of medicine should stress the importance of stated regular physical examinations.
2. In early diagnosis rests the secret of operative success in cancer of the breast.
3. The proportion of benign tumors of the female breast is greater than is generally quoted.
4. Removal of breast with thorough cleaning out of all glands is often, but not always, justifiable in advanced cases.
5. Eventually a cancer cure wlil be found.

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DISCUSSION

DR. J. R. VAN ATTA, Albuquerque, N. M.: It has occurred to me that the family history might enter into these cases. A woman came to me with an extremely bad cancer history, all of her sisters and aunts on her mother's side having had cancer. I removed her right breast with radical operation. Just exactly one hundred days from the first operation, she returned and said she had a stinging pain in the opposite breast; that breast was removed on the basis of this stinging pain and a nodule whcih you could not satisfy yourself was present, and it was found to be carcinomatous. I felt chagrined that I had not elicited some evidence of the involvement of the other breast when only a hundred-day period elapsed bteween the two operations. She came to me the first moment she discovered the nodule. She knew the value of early diagnosis. The principal point is that there was apparently a hereditary taint in that family. The history is very important and I think as much so as the physical examination.

DR. R. O. BROWN, Santa Fe, N. M.: This reminds me of Maude Slye's work with white mice. It seems to me it contains a lesson for every physician in the country to be sure and investigate the family history for cancer, especially when there is any suspicion of cancer in the breast, as, beyond question, there is a strong hereditary influence.

DR. BAKER (Closing): In a paper of this kind it would take too much time to discuss the various and different phases which we should enter into upon making reports on tumors of the breast, but we should all realize the importance of taking careful histories in these cases, as Dr. Van Atta has brought out. About twenty years ago, there were published cancer abstracts and you could see the number of cases of cancer in your records, where reported cancers had been found among the members of a family.

I wish to thank the New Mexico Society for a wonderful time and hope I may have the pleasure of meeting with you again.

HYSTERECTOMY. A TECHNIC ANALYSIS OF 604 CASES

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(Read before the New Mexico Medical Society, at its forty-eighth Annual Meeting, at Raton, N. M., June 4-6, 1930.)

INTRODUCTION

Since Atlee reported the first hysterectomy before the American Medical Association in 1853, numerous articles have appeared in the literature concerning its technic and indications. It is only in the last two decades, however, that such writers as Kelly, Cullen, Mayo, Peterson and Lynch have reported their complications, morbidity and mortality. While a surgeon's recoveries greatly outnumber his fatalities, there is considerable value in placing these on record.

Our chief concern should be to keep mortality at a minimum. We must also endeavor to reduce our complications and improve surgical convalescence. Polak, and one of the writers (DGT) have shown that the preoperative analysis of the patient's condition, avoidance of multiple operations, will reduce morbidity and mortality. The type of work of Dr. Peterson at Ann Arbor, in classifying gynecological risks, is an important contribution to surgery, as well as an essential to success.

Indication for the removal of the uterus should be greatly reduced with the advent of radium and x-ray; for endometrial hyperplasia, small fibroids, and the so-called "fibrosis uteri" are well handled by this form of treatment. Soiland, Costolow and Meland have recently reported 562 cases treated by radiation, with no mortality, and with only seventeen requiring subsequent

removal. A word of warning, however, may be sounded in this regard, as the contraindications to the use of this form of treatment are definitely defined and surgery is still the choice of treatment in the large majority of bleeding uteri.

Conservation of the uterus should be practiced by all surgeons, for in spite of the ruthless sacrifice of the organ because of insignificant pathology or disease of the tubes, no one has yet shown that the muscular structure, which is the seat of the menstrual response and the protective cavity for the developing fetus, does not have some other function in the physiology of the female. Removal of the uterus must be done only when the weight of the evidence indicates that its extirpation is both beneficial and a necessity.

In analyzing 604 hysterectomies, we have attempted to present our findings in four divisions, as follows.

1. The preoperative status of the patient
2. The operation
3. The complications and morbidity
4. The immediate mortality and convalescence.

PART 1.

The Preoperative Status.

The ages of all types of cases are listed in Table 1:

Table 1.—Age Incidence.

20-29	years	6.0%
30-39	"	35.6%
40-49	"	45.3%
50-59	"	10.0%
60-69	"	1.8%
70-76	"	0.4%

Youngest, 20—oldest, 76.

These percentages correspond quite closely with those of Lynch in his series of 683 fibroids. It will be noted that the largest percentage of patients developed uterine pathology in the late thirties and forties.

Marital State and Pregnancies.

Forty-four patients had never been married, while in twenty-five this point was not stated. Of the remaining 535, eighty-one were sterile, forty-two had been pregnant, but had no children, sixty-six had one child and fifty-eight had two children. Disease of the uterus naturally lowers the fertility of the individuals. However, in over half of the cases, the uterus was not removed until the usual child-bearing period was past. In many instances patients had been followed for several years before operation was performed and some never required surgery. Fibroids without symptoms need watching, not surgery.

Symptoms.

Eighty-nine patients of the 590 complained of menorrhagia or metorrhagia or both. In 197, bleeding was associated with enlargement, tumor, pain, or miscellaneous symptoms. Disease of the uterus is indicated by hemorrhage from the endometrium in nearly half the cases. Pain was given as the only symptom in eighty-three. The complete resume of the symptoms is tabulated in Table 2.

Table 2.—Symptoms

Hemorrhage		89
Menorrhagia	42	
Metorrhagia	18	
Both	29	
Hemorrhage and pain		80
Hemorrhage and enlargement		16
Hemorrhage and miscellaneous		101
Pain		83
Pain and miscellaneous		74
Tumor or enlargement		34
Miscellaneous		86
Vaginal discharge		10
Prolapse		9
Not classified		14

Miscellaneous refers to symptoms of the gastro-intestinal, genito-urinary, or neuromuscular systems.

Our routine procedure calls for complete history, physical examination, urinalysis, blood count, and serology. Physical examination revealed five patients with active tuberculosis. There were four definite cardiac lesions, with numerous references to poor myocardium and low cardiac reserve. Thirty-eight patients had a systolic blood pressure of 160 mm. of mercury or above. There were eight diabetics. One hundred patients showed a trace or more of albumin in the urine, twenty-five showed casts to be present, and in thirty-one the urine contained a large number of pus cells. There were nineteen patients with 4 plus Wassermanns. There were seventy-seven patients who had a hemoglobin below 60 per cent. There were also seventy-seven who had a leukocytosis of 11,000 or more. There were, therefore 306 patients who showed some factor which would place them in the class of poor surgical risks. Twenty-three of the anemic patients were transfused. While it is generally accepted that patients should not be operated upon with an elevation of temperature, twenty-four of this group were operated upon with temperatures of more than 99 degrees. All cardiacs, nephritics, and diabetics were hospitalized for from three days to a week before operation was performed. This has been found to be a great asset in improving the surgical risk—while the average hospital has but little authority in saying who can or cannot operate, for the protection of the patient there should be a minimum period of forty-eight hours of observation on all elective surgery. Two days' rest before operation would be more advanta-

geous than the thirteenth or fourteenth day postoperative in the hospital.

The cry for reducing hospital expense is disinctly increasing the danger of hurried elective procedure, and, unless controlled by surgeons, dangerous complications will result. Over 85 per cent of the patients operated upon in this group have been observed at least three days before admission for surgery. During this time the laboratory tests are made, incident to a complete study, and the patient is then accepted or rejected for immediate operation.

Previous operations further increase the risk of surgery. The adhesion and scar tissue make the operation more difficult, and foci of infection may be activated. A classification of previous abdominal operations is shown in Table 3.

Table 3.—Previous Operations.

Number of patients having previous laparotomies	120
" " pelvic operations	36
" " pelvic operations and appendectomy	39
" " appendectomies alone	35
" " previous upper and lower abdominal operations	6
Miscellaneous	4

Two patients had had nephrectomies and, in summarizing, it will be noted that 79 9patients had their appendices out before operation for hysterectomy.

PART 2.

The Operation

There were 527 subtotal hysterectomies, thirty-six total and four vaginal hysterectomies. Thirty-seven were called "hysterectomies" and are not classified. Their pathology and type of operation are tabulated in Table 4.

Table 4.—Pathology.

	Supracervical		Total		Not Class.		Totals	
	Recov.	Died	Recov.	Died	Recov.	Died	Recov.	Died
Fibroid	364	6	17	1	23	0	404	7
Fibrosis-metritis Endometritis-hyperplasia	51	2	3	0	3	0	57	2
Adenomyoma, uterus	14	0	0	0	1	0	15	0
Salpingo-oophoritis	21	0	3	0	3	0	27	1
Endometritis, acute	0	0	1	0	0	0	1	0
Carcinoma, uterus	7	0	5	1	1	0	13	1
Carcinoma, cervix	0	0	2	0	0	0	2	0
Carcinoma, polyp	0	0	0	0	1	0	1	0
Carcinoma, ovary	4	0	0	0	1	0	5	0
Carcinoma and fibroid	6	0	0	0	0	0	6	0
Sarcoma of fibroid	1	0	0	0	0	0	1	0
Interruption of preg.	1	0	1	0	1	0	3	0
Incomplete abortion	0	0	0	0	1	0	1	0
Pregnancy and fibroid	19	0	0	0	1	0	20	0
Polypi	8	0	1	0	1	0	10	0
Normal	7	0	0	0	0	0	7	0
Not stated	15	0	0	0	0	1	15	1
TOTAL	518	9	33	3	37	0	588	12

4 vaginal hysterectomies—all for prolapse; uterus normal in 3, fibroid in 1.

The technic that will be presently described is the subtotal type. The operations, with eight exceptions (spinal), were done under ether or gas-ether anesthesia. In only a very few instances was there any preoperative opiate. The advantage of ether lies in its safety, ease of administration after establishment, and the more perfect relaxation. It has a certain disadvantage which will be later reviewed.

After perfect relaxation, the patient is placed in the Trendelenburg position. The second most important step is an adequate incision. In nearly all cases, the incision extends from the symphysis to the right and slightly above the umbilicus, avoiding this fibrous structure. The little difference in the length of the incision should not be considered when it is of such unquestionable value in exposure without the presence of another pair of hands in the vicinity of the wound.

The corkscrew is then used to deliver the fundus and allow traction on the ligaments and bladder reflection. The round ligaments are then clamped close to their fundal insertion, and an incision made with a knife on the peritoneal reflection extending from side to side. This flap of peritoneum is then pushed down with a piece of gauze in thumb forceps, freeing the uterus in front. The broad ligaments are then double clamped and severed, pointing the clamp inward toward the body of the uterus. This usually exposes the uterine vessels, which are clamped close to the uterus with as little tissue as possible. With a knife, the uterus is then dissected from the cervix. A traction suture is now placed on either side of the canal, using No. 2 chromic catgut, tied, clamped, and held without removing needle. The cautery is then plunged deeply into the cervical canal about one centimeter in diameter and left open. The uterine vessels are then tied separately with double No. 2 chromic catgut. The broad and round ligaments are similarly treated, anchoring suture at both ends of the broad ligament to prevent retraction of contained vessels. The traction cervix suture on the left side is then opened to allow the left round and broad ligament stump to be brought into its grasp and further anchored by the needle in this suture. The opposite side is similarly treated, which supports the stump. The peritoneal flap is then brought over the contained stump, and all raw areas are properly covered with this flap. The cervical canal has then one layer of peritoneum over it, and the tissue distal to the tie which has an outlet through the vagina. This, we believe,

is an important step in reducing the morbidity in hysterectomy.

While no claim is made for the originality of the operation, the technic herein described differs in the treatment of the stump from that usually described in textbooks.

The presence of existing pathology in other organs will also increase the complications of uterine surgery. The routine examination of the upper abdomen revealed sixty-two gallbladder lesions, showing the importance of this palpation in elective surgery. The remaining pathology as listed was incident to the condition for which the uterus was removed. In 101 adnexal infections, twenty-eight uteri were removed because of generalized inflammatory process of the pelvic viscera, making the uterus a foreign body when no ovarian tissue was left.

Table 5.—Associated Pathology.

Gallstones	35
Cholecystititis	22
Distended gallbladder	4
Papilloma, gallbladder	1
Duodenal ulcer	1
Colitis	1
Acute appendicitis (ulcerative)	1
Adnexal infection	101
Ovarian cysts, simple	35
Ovarian cysts, multiple	42
Ovarian cysts, dermoid	9
Corpus luteum cysts	3
Cystadenoma	4
Pseudomucinous cyst	2
Adenofibroma, ovary	1
Teratoma, ovary	1
Ectopic pregnancies	2

PART 3.

Postoperative Complications and Morbidity.

In the 315 hospital charts analyzed, there were ninety patients who apparently had symptoms suggestive of head colds, acute coryza, or chronic respiratory disease. There were two definite broncho-pneumonias, eighteen who had bronchitis, and seventy who had tracheitis, pleurisy, pain in the chest, and received some type of treatment for this complication. This is definitely one of the troublesome complications, or the one disadvantage of ether inhalation.

There were seven cases of thrombophlebitis. Surgical shock (severe) six. Gastric dilatation, seven. Postoperative cervical hemorrhage, two. Vesicovaginal fistula, one (healed spontaneously).

The large incision may increase the possibility of infection. Thirty-nine patients had wound infections. A very troublesome complication is serum in wounds. One hundred and seven patients had such complications. There was only one wound rupture which was resutured with no untoward symptoms.

A patient was considered to have morbidity when the temperature was 100.4 on any two days not including the day of operation. One hundred and seventy were morbid. Of these, fifty-four had more than three days.

Our morbidity was apparently largely due to wound infection, serum and pulmonary complications. Early in the series, the technic of the operation was responsible for a number of severe parametric infections and peritonitis. The drainage per vagina, we believe, is a definite advantage. On the fourth to sixth day, the patient had a discharge of necrotic material from the stump, which is the result of the cauterization, and the material distal to the ligatures under the peritoneal reflection.

PART 4.

(A) Mortality

There were twelve deaths in 604 cases, or a mortality of 1.9 per cent. In analyzing deaths, we must consider the preoperative status, the operation, surgical accidents, and postoperative complications.

The summary below shows three total hysterectomies, nine supracervical. Note that in seven cases there was evidence in the preoperative study that the patient was not a good risk. In one, surgical shock from hemorrhage resulted in death. Malignancy of the uterus in a woman of 76 may be classed as a poor surgical risk. In the remaining two, both developed mechanical obstruction and peritonitis, one had had a previous operation for pelvic inflammation, in the other appendectomy was done in addition to the pelvic operation.

In 311 hysterectomies performed in Peterson's Clinic, seventeen of which were radical panhysterectomies for cancer of the cervix, the mortality was 4.5 per cent and one half of the fourteen deaths were panhysterectomies for existing pelvic inflammatory disease. In our series of deaths, disease of the adnexa was present in six, three of which developed peritonitis.

Case 1. No. 4719, age 40, albumin in urine; leukocytes, 12,500, polys 90 per cent. Subtotal hysterectomy, bilateral salpingectomy and oophorectomy for fibroid and cystic oophoritis. Operation required one hour. Developed obstruction 4 days after operation. Reoperated on 6th day. Died 16 hours after operation to free adhesions between bowel and wound. Believed low grade infection and peritonitis caused by obstruction.

Case 2. No. 6098. Total hysterectomy for cervical fibroid—required two hours to remove. Patient was badly shocked, due to severe hemorrhage, died same day, possible clamped ureter, shock due to hemorrhage.

Case 3. No. 6990, age 51, hyaline casts and albumin in urine. Total hysterectomy for fibroids and repair of umbilical hernia, required 64 minutes. Iodoform gauze in vagina. Died in three days, of peritonitis.

Case 4. No. 9126, age 76. Total hysterectomy for adenocarcinoma. Died two weeks after operation, cause not stated.

Case 5. No. 15421 age 41. Trace of albumin and casts in urine—B.P. 234/140. Subtotal hysterectomy and right salpingo-oophorectomy for fibroid uterus. Died 19 days after operation; coma 36 hours, urea 25 mg. Chronic nephritis, chronic cholecystitis, chronic myocarditis, fibroid uterus.

Case 6. No. 14296, age 33. Temperature 99. Supracervical hysterectomy and right salpingo-oophorectomy for intraligamentous fibroid of uterus and hydrosalpinx; 82 minutes, considerable hemorrhage. Died 41 hours after operation, 1½ hours after gastric lavage. Possible ligated ureter. Cause of death, peritonitis.

Case 7. No. 20356, age 41. Temperature 102.6; pulse 124, leukocytes 26,800; polys 91 per cent. Supracervical hysterectomy and bilateral salpingectomy and appendectomy and drainage; 100 minutes. Hydrosalpinx, pyosalpinx, fibromyoma, acute endometritis, chronic appendicitis. Died 6 days after operation; peritonitis.

Case 8. No. 10618; age 38. Positive Wassermann, albumin in urine, pus cells in urine. Subtotal hysterectomy and perineorrhaphy for submucous fibroid and adenomyoma. Died 7 days after operation, of postoperative peritonitis.

Case 9. No. 25067; age 26. Faint trace of albumin in urine. Subtotal hysterectomy and oophorectomy. Previous operation for inflammatory disease. Died 8th day postoperative, of peritonitis—mechanical obstruction.

Case 10. No. 16022; age 43. Hemoglobin 41 per cent; leukocytes 9000; polys 80 per cent; temperature 99.4 before operation. Transfused, subtotal hysterectomy for submucous fibroid and adenomyoma. Wound infection. Died 11th day postoperative, peritonitis and sepsis.

Case 11. No. 27711; age 48. Temperature 99.4 before operation; cough. Subtotal hysterectomy, left salpingo oophorectomy, and appendectomy. Distention; vomiting; cough and cardiac. Died 9th day postoperative, of paralytic ileus.

Case 12. No. 12913. Endometritis. Subtotal hysterectomy and appendectomy. Curettage 4 days before. Explored for obstruction on 9th day. Died 11th day postoperative, peritonitis.

PART 5.

(B) Follow Up.

In the total of six hundred and four cases, sixty-four were not heard from after operation. Seventy-four patients had no relief of symptoms. Fifty-two patients complained of bladder symptoms. The operation might possibly be responsible, but in analyzing the hospital charts we find many patients whose bladders became overdistended. The fear of catheterization is so instilled in the nursing profession that many patients have the greater danger thrust upon them—bladder distention. The percentage of cystitis caused by faulty catheterization is very small, while overstretching of the bladder mucosa, plus stagnant urine, causes the large majority of these troublesome complications. The order of catheterization as necessary should be changed to every eight hours, and once after voiding for residual urine. Here, again, the frequent voiding of small amounts is rarely diagnosed as an overdistention, although this is a common nurse's report on a chart.

The second most common follow-up complication is vaginal discharge. Subtotal hysterectomy per se will not cure an infected cervix. Fifty-three patients returned with vaginal discharge as their chief distress.

In this series not a single incidence of carcinoma of the retained stump was brought to our attention. Two cervical polyps developed, and one fibroid of the cervix, but in a relatively large series the cauterization may have been of some benefit.

In forty cases, the cervix was removed by the operation; in ten, trachelorrhaphy was used to reduce the size and repair the lacerated structure; in four, a partial amputation was done; in none of these return of this symptom. In eleven per cent, therefore, the operation did not remove one of the symptoms for which the surgery was performed.

Prolapse of the stump occurred in four instances. Three of these occurred early in the series and before the careful suspension with the round and broad ligament was used.

Five patients developed postoperative ventral hernias, which were repaired. In three instances, ovarian cysts developed in the retained glandular tissue. Eight of the diseased gallbladders with stones, which had been diagnosed six years before. One of the 279 retained appendices became acute and required operation.

As previously stated, seventy-four patients were not improved. In this group, the symptoms were pain, backache, and symptoms in associated organs. Most of the uteri in this group were classed as metritis, fibrosis and normal. The removal of the uterus will not cure a large number of these cases because the underlying pathology was apparently elsewhere.

Ten patients died, according to our follow-up: two diabetic, two malignancies, one tubercular. One patient developed an obstruction a year later and died following operation, one developed a brain tumor, one committed suicide, and in two others the cause was not known.

Of the total group, 140 patients had menopausal symptoms, or an evidence of 26 per cent. Symptoms occurred in 12.6 per cent where both ovaries were retained; 25.5 per cent where one ovary was left. When both ovaries were removed, 36.3 per cent had menopausal symptoms.

The following tabulation is of interest:

Table 6.

	Under 45 yrs.		45 yrs. and over.	
301 Hysterectomies-ovaries retained	44	14%	15	5%
176 Hysterectomies-one ovary removed	34	19%	11	5%
96 Hysterectomies-both ovaries removed	16	16%	20	20%
Total	94		46	

7 Transplants 3 had symptoms

4 Radium or x-ray before operation—no symptoms

A greater attempt should be made to conserve ovarian tissue. Endocrine products are satisfactory and should be used, but many patients are not relieved of these distressing symptoms by glandular therapy.

CONCLUSIONS

1. Hysterectomy in all types of cases should not carry a mortality greater than two per cent. In some series of fibroids it should be considerably less.
2. Even in the most careful aseptic technic, evidence of wound infection will be high because of the frequent existence of low grade pelvic infection.
3. Careful preoperative study and improving the general condition of the patient is more important than the rapid, carefully executed hysterectomy.
4. Ovaries should be retained whenever doubtful disease exists.
5. The cervix should be cared for at the time of operation.
6. Miscellaneous symptoms and palpable tumors should not be indication for operation.

DISCUSSION

DR. W. J. LATTA, Wagon Mound, N. M.: I think perhaps I saw the first hysterectomy that was successfully performed west of the Allegheny Mountains, in 1874, and by a doctor who had very little experience in these things. The woman had a fibroid which bled very freely. In those days they did not know much about radium. The woman made an uneventful recovery and I saw her forty years later (she was 30 years old at the time of the operation) and asked her about herself, and as to the effects of the operation. She replied that it made such an enormous change in her mentality and general feeling that she would rather have died than not had the operation performed.

DR. P. G. CORNISH, Jr., Albuquerque, N. M.: I am very much interested in the analyses of these hysterectomies. The mortality and morbidity are very low. It seems to me that in hysterectomies, the percentage of wound infections—and there is quite a large percentage in this series—is probably due to infection in the cervix. In technic where the cervix is left open quite a long time, I should not be surprised if that was the cause of the wound infection. Personally, I like to get the cervix closed as soon as possible after it is cauterized. I do not know that there is any particular advantage in leaving it open. Dr. Tollefson's paper is certainly a very interesting analysis of a great many cases, showing excellent results.

DR. TOLLEFSON (closing): In regard to the cervix, I had never seen this same thing done in my training. The reason Dr. Moore changed to this technic was that quite a few would have an elevation of temperature about the fourth, fifth or sixth day. In such cases, he would stick a clamp up into the cervical canal, establish drainage, and the temperature would come down. Leaving the cervix exposed is probably one of the factors in wound infection. However, every attempt is made to keep anything threatening away from low-grade infections existing in the pelvis.

I wish to express my appreciation and thanks to the Society for inviting us to read this paper.

PHYSIOLOGICAL AND CLINICAL ASPECTS OF KIDNEY DISEASE

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(Read before the New Mexico Medical Society, at its forty-eighth Annual Session, at Raton, N. M., June 4-6, 1930.)

The concept of kidney disease has undergone considerable change during recent years. Professor A. N. Richards has devised methods to give us better knowledge of the normal function of the kidneys. Chemical studies of the blood and body fluids; detailed histologic studies of the diseased kidney; routine use of the blood-pressure apparatus and of the opthalmoscope have added to our understanding, and today we have a clinical concept of renal insufficiency which is somewhat different from that held by the older physician.

FUNCTION OF THE KIDNEYS

The kidney is the chief excretory organ of the body. Its function is to excrete water, to excrete waste substances of body metabolism, urea, uric acid, creatinin, and to maintain an acid base balance for the body economy. When an excess of acid is present in the body, base is retained by the elimination of acid phosphate. The regulation of water content is by far the most fundamental and most sensitive function. Cushney places the maximum capacity of the normal kidney to excrete water as one liter per hour. Cases of diabetes insipidus have been reported in which twenty liters of urine have been eliminated in twenty-four hours. Frequently cases are observed where an intake of one liter of water is followed within two and one half to three hours by its total excretion as urine. Diuresis usually begins in one half to three quarters of an hour. This functional ability of the kidney to excrete an excess of water is made use of clinically by the so-called "water test" of Volhard. Fifteen hundred or, preferably, 1000 c.c. is taken on an empty stomach and its time of elimination observed.

The ability of the kidney to prevent the elimination of water is equally efficient. In hemorrhage, severe diarrheas and dysenteries and in profuse sweating, the extreme loss of water may go to the complete suppression of the urine. Under normal conditions, however, the urine is concentrated: only sufficient water eliminated to keep in solution the waste substances being eliminated. So we may measure this concentrating power by two-hour determinations of the specific gravity.

Our knowledge of the manner in which the kidney performs this function has been greatly enlarged by the work of Professor

Richards. The glomeruli are tufts of arterial capillaries. As the blood passes through them, a deproteinized solution containing the other constituents of the blood plasma passes out of these capillaries and the covering membrane of Bowman's capsule. This enormous capillary surface of the kidney is not always open in its entirety for the passage of blood, for if the capillaries were all open, a kidney excreting one half a liter an hour would not be able to excrete half a liter in twenty-four hours. Richards has shown that only a limited number of these glomeruli are open capillaries at one time, and that these glomeruli open are dependent upon the amount of water the kidney has to excrete. He has further shown that the activity of a single glomerulus may vary. When the protein free solution has passed out of the glomeruli, it passes through the lumen of the convoluted tubules, the cells of which absorb the substances, water, sugar, salt and base which the organism needs in its economy.

In the excretion of waste substances of protein metabolism, the kidneys are almost alone concerned. Urea, uric acid, creatinin and some other undetermined substances of the total nonprotein nitrogen of the blood will be excreted by the kidney until two-thirds of the kidney substance is destroyed. When more than two-thirds of the kidney is out of commission, then only will these waste substances be retained in the body. When kidneys are able to excrete an excess of water in a certain minimum period of time, when they are able to concentrate the urine to a certain maximum degree; when they are able to eliminate waste substances under all possible conditions of natural protein intake, they are normal efficient kidneys; when they cannot do this, they are spoken of as insufficient.

The loss of function of excreting waste substances being the last to be manifest in kidney structure loss, it follows that retention of nonprotein nitrogen represents an absolute insufficiency, and this may be measured by the amount of the nonprotein nitrogen and creatinin in the blood plasma.

Between the normal functioning kidney and the absolute insufficient kidney indicating two-thirds destruction of the organ, there is a wide range. All grades of lengthening of time of water excretion, all grades of diminution in the ability of the kidney to concentrate the urine, occur with kidneys still able to excrete waste substances. When the highest specific gravity of the urine cannot go above 1018, one knows the kidneys are relatively insufficient to that degree. When the ability to concentrate is at its lowest ebb, the urine remains fixed at about 1010 to 1012, and its concentraion is about the same as the molecular concentration of the protein-free blood plasma. The tests, therefore, for determining the relative insufficiency are the water excretion test and the two-hour concentration test. The tests for absolute insufficiency lie in the above tests with the chemical examination of the blood for retained nonprotein nitrogen. The dye test of kidney function has not maintained its early clinical importance, excepting in the hands of the urologist, as a rapid measure of separate kidney function. Under a wide range of conditions, phenolsulphonepthalein and indigo carmine may show marked variation, and the range of 40 per cent to 60 per cent of the dye being excreted restricts its use to an indicator of the completely inefficient kidney.

CLINICAL RENAL DISEASE

The changing clinical symptoms of kidney disease often lead to confusion in interpreting the renal lesion or the disease.

One or more of the following clinical signs are always present in kidney lesions:

1. Urinary findings.
2. Edema.
3. Blood pressure—hypertension.
4. Nitrogen retention—blood chemistry.
5. Opthalmoscopic findings.
6. Uremic.

Every physician has struggled to interpret one or more of the above symptoms into terms of the actual kidney lesion. In the nephritides, in the nephroses, in the hypertensive states, in arteriolosclerosis, and atherosclerosis, in cardiovascular breakdown, these clincial signs present themselves. We have familiarized ourselves with various classifications of nephritis at the present time—acute nephritis, subacute nephritis, subchronic nephritis and chronic nephritis have become an excellent working classification. Distinguished from them, however, we now have the condition called "the nephroses."

NEPHROSES

In 1905, Mueller used the term nephroses to designate tubular degeneration of the kidney in distinction to the term nephritis, or glomerular inflammation. In 1914, Volhard and Fahr in a monograph used this term to classify clinically cases in which there was no evidence of inflammation of the glomeruli at autopsy, but still present clinical evidence of renal disease; albuminuria and edema. The pathologic changes in the kidney included all forms of tubular degeneration and necrosis. At the present time, nephrosis is used in a pathologic sense. Clinically, in febrile diseases, we find albu-

minuria, and histologically, cloudy swelling of the tubules. To speak, therefore, of a nephrosis as the urinary finding of a pneumonia, or a typhoid, is to raise an important clinical symptom to the dignity of a disease. Nephrosis may, however, justifiably be used as a term to define a group of kidney lesions that are well defined: bichloride poisoning, kidney of pregnancy, amyloid disease and lipoid nephrosis.

LIPOID NEPHROSIS

Volhard revived the term nephrosis to classify this definite clinical syndrome: oliguria; marked albuminuria; marked edema; paleness of the skin; increased lipoids and cholesterin in the blood; absence of hematuria, hypertension and cardiac hypertrophy; and without evidence of renal insufficiency.

I am at the present time observing five cases in which this diagnosis of lipoidal nephrosis is still under question. In all the cases, the disease began insidiously, and has run a chronic course. The severe albuminuria in each case has reduced the albumin portion of the blood plasma with a relative increase of the globulin portion and repeated readings of from 3 to 10 grams of albumin per liter are obtained. Oliguria from day to day is usually present. In two of the cases, the cholesterin of the blood, which normally should be 0.21 per cent, is increased to .5 per cent and .72 per cent respectively. Fatigue and pallor are a constant observation. In two cases the first symptom that drew attention to the illness was edema of the ankles. The specific gravity may be very high. In my study, hyalin and granular casts and lipoidal bodies were found. Two-hour concentration tests show no fixation. Blood pressure remains normal, and basal metabolic rate is uniformly reduced to -15, -20 in all cases of these five. It must, however, be stated that this clinical entity of lipoid nephrosis may appear as a phase of acute glomerulo-nephritis as well as a separate clinical entity. Murphy recently reported a case of chronic glomerulonephritis showing at one time all the characteristics of a lipoid nephrosis. He observed this case for a period of five years. As the disease progressed hypertension and renal insufficiency ensued.

When the symptom complex of lipoid nephrosis is found without evidence of nephritis, one is unable to tell whether one is dealing with a case of pure lipoid nephrosis or with the symptom complex occurring as a tranistory phase in the course of a nephritis. One case—a man 20 years of age, with the gradual onset of fatigue, edema of ankles and eyelids, pallor, slight subcutaneous edema, basal rales, oliguria, albuminuria, casts, no hematuria or blood cells in the urine, normal blood pressure and kidney function and lipoidal bodies in the urine with hypercholesturemia; all of these characteristic manifestations of lipoidal nephritis—has further evidence of advanced pulmonary tuberculosis. The question immediately arises whether this, perhaps, is not a case of amyloid nephrosis.

The nephrosis of pregnancy, or mercuric chloride poisoning, amyloid degeneration of the kidney, must be differentiated and classified on a basis of its etiology.

GLOMERULONEPHRITIS

The changeableness of the symptoms of chronic nephritis often leads to confusion in the proper interpretation of the clinical picture in some stages of the disease. Lohlein has shown that all forms of nephritis have their beginning in an acute diffuse inflammation of the glomeruli. He further showed that all of these cases of glomerulonephritis that are of longer standing than the acute form, fall into three groups. One form has a short stormy course with edema, marked changes in kidney function, albumin and blood cells, granular and fatty casts, early blood retention with increase in blood pressure, and eye-ground findings. These cases may well be classified as subacute glomerulonephritis. They have a fatal termination within a few months. In the second group, the character of the disease is milder and lasts from a few months to one, or even two years. They correspond to the subchronic form of glomerulonephritis. The third group run a very chronic course over many years. Both the second and third groups may be further divided on the basis of whether they show edema. They are easily confused with cases having cardiovascular degenerations and cardiac breakdown, high blood pressure, nocturia, fixation of the specific gravity, lowered phenolphthalein output with, as a rule, slight trace of albumin and other minor evidences of inflammation of the glomeruli. These cases are subject to frequent acute exacerbations, and occasionally periodic hematuria. The nonprotein nitrogen gradually rises as absolute insufficiency takes place, and uremia closes the picture.

BENIGN AND MALIGNANT SCLEROSIS

Another modern clinical concept of kidney disease is the cases of benign and malignant arteriolosclerosis with and without renal insufficiency. Volhard demonstrated the obliteration of a greater or lesser number of the glomeruli of the kidney without inflammatory changes. These show a chronic hypertension, variable to late middle life, when

they become permanent hypertensive cases. They are familiar to us all under the name of "hyperpiesia or benign hypertension." Dyspnea epistaxis, high blood pressure for many years, left ventricular preponderance, are the prominent symptoms. The urine shows a trace, or no, albumin, hyalin casts, some impairment of renal power to eliminate fluids: but these cases do not die of renal insufficiency, but of intercurrent infections, of diseased coronary arteries, or arterial accidents of the brain.

It is noteworthy that among the group of chronic hypertensive cases there is a small group in whom, after prolonged hypertension, profound renal insufficiency will supervene. These cases are the malignant renal sclerotics. Many of these cases are being reported. Without discussing the histological process of its production, the kidney gradually fails in its function, nitrogen retention supervenes, and gradually becomes more pronounced. Clinically these are cases of hypertension of many years' standing, in which is gradually added renal insufficiency; inflammatory changes in the kidney being absent.

In conclusion, the purpose of these remarks is to point out the importance of some of our present methods of clinical diagnosis of renal impairment, and to sketch a working classification for the study of kidney disease.

DISCUSSION

DR. R. H. FINNEY, Pueblo, Colo.: This is a very interesting subject. There has been so much work done in the past ten years in trying to work out a classification of kidney diseases that it makes it most interesting in physiology. I certainly do not want to say one word that would take your minds away from this excellent physiology Dr. Heller has blocked out, and I do not intend to get away from that line, but for general practitioners let us call any of these diseases Bright's disease, rather than to say this is Bright's disease, this is nephritis, and so forth. Let us make it as simple as possible and say Bright's disease. However, there is more than one disease in the kidney function. Dr. Heller has brought out the physiology up to the present time, as much as is known about it. There is a great question as to what nephritis is and where it belongs in the classification. When it comes to making a diagnosis, putting down our preliminary diagnosis and final death certificate diagnosis, I try to come down to one of three:

(1) Hemorrhagic nephritis in which there is an acute condition—first, regardless of how it gets later. Some of them clear up never to return again. In that case there is blood and very little else, unless perhaps a little albumin. It may be from an acute inflammatory condition, scarlet fever, or some streptococcic infection.

(2) Degenerative nephritis, which is very broad, but takes in all degenerative conditions. In this, degenerative debris is found in the urine, but no blood, and rarely red blood cells. It might include Bright's disease of pregnancy and there are many others so intermingled with this degenerative type, I usually call them degenerative nephritis and let it go at that.

(3) Arteriosclerotic nephritis in which some albumin, sometimes some little hypertension and arteriosclerosis changes are found throughout the kidney. There are no red blood cells and no degenerative cells to amount to anything. These are the three types used by me in a diagnostic standpoint.

DR. J. F. PERCY, Los Angeles, Calif.: I should like to ask Dr. Heller if he has made any study in regard to the relationship of lipoidal nephritis to the so-called nephritis of Epstein, and whether or not these two conditions bear any relationship to each other. I am really interested in this because of some work that I did years ago. I published two papers, one in 1912 and the other in 1913, in reference to the relationship of the thyroid to changes found in the kidney. It is an exceedingly interesting subject.

The best treatment for such cases is the administration of very large doses of thyroid extract. In my own mind the nephritis of Epstein is nothing more nor less than what has been mentioned under the general term of nephritis. The excessive amount of albumin in the urine in these cases is most remarkable—in fact, the urine of all these individuals is really a menagerie. I remember one specimen I kept for many years and exhibited it, as you could find there anything that you wanted. Many of these cases, however, absolutely clear up and get well through the administration of thyroid extract. My attention was first directed to this condition because of a patient I had who had an enormous cystic goiter—a man. He got entirely well under the administration of thyroid extract.

DR. HELLER (closing): In regard to the nephritis of Epstein, he classified all nephroses together and the separation is taking place at the present time, since his publication. To say that this or that case is lipoid nephritis is a very difficult task just now, but such cases are being found. They are going along much as Dr. Percy has indicated. With thyroid treatment many are remarkably improved, even being discharged as well, in a number of instances. Other cases so treated, however, show no improvement.

The interesting feature under study at the present time is to decide whether the process of nephrosis is one in which pneumococcus is a contributing factor.

I am very glad that Dr. Percy brought out the matter of treatment, because the treatment of lipoidal nephrosis is still in an experimental stage. Treatment of lipoidal nephrosis of pregnancy is being tried with thyroid extract. As to the problem of uremia, I think this subject is tremendously misunderstood. We have not arrived at a very wonderful point in a better chemical and physiological knowledge of this process. There are some who believe that there is a toxic element in the production of clinical manifestations of uremia; others believe it is simply an enormous accumulation of urea in the body, which produces it. Many of us think it can be a convulsive manifestation; others, that it can be simply a twitching and that the convulsion is not a necessary part of the disease. They are due to an opacity of the arteries of the brain and are not produced by the poison that produces the clinical manifestations of uremia. The stupor, respiration, and so forth, is a part of the picture. As to treatment, I firmly believe that sugar and water in every way possible are of advantage in many cases of uremia.

I have wanted to give you this from a physiological standpoint to the practitioner. Remember that your tests on kidney insufficiency or impairment are: (1) water elimination; (2) the power of the kidney to concentrate its urnie. A kidney that can not concentrate above 1018 is a partially incompetent or insufficient kidney. A kidney that cannot concentrate its urine above the point 1012 is a badly insufficient kidney and you will find those tests to be of much value to you.

"POTPOURRI"

W. L. BROWN, M. D., and C. P. BROWN, M.D.
El Paso, Texas.

(Read before the New Mexico Medical Society, at its forty-eighth Annual Meeting, Raton, N. M., June 4-6, 1930).

Practical Points in:

A. EXOPHTHALMIC GOITER.

Exophthalmic and toxic goiter cases are still reaching the surgeon, after they have tried all the other varieties of treatment, with cardiac decompensation, a severe degree of thyrotoxicosis, accompanied by swollen extremities, high degree of nervousness, tremor and enormous exophthalmus. We think the great majority of these cases could have been saved from this most serious condition, if they had been sent to the surgeon at a very much earlier date. Of course, practically all of the exophthalmic cases will respond to sufficient doses of iodine, but it must be remembered that iodine is not curative and the danger is that it may be used over a period of months or years and may finally lose its effect.

Now that iodine is found to be specific in temporary control of exophthalmic goiter symptoms, another confusion has arisen and that is the routine prescribing of iodine in goiter cases regardless of their nature. It seems well established that iodine in the ordinary non-toxic adenoma of the thyroid may cause it to become toxic. Therefore it appears that iodine should be reserved, first, for the control, not cure, of symptoms in exophthalmic goiter; second, there is probably no harm in giving it to the already toxic adenoma, as occasionally there are mixed types and some benefit may be derived; third, it is often of value in the adolescent or endemic goiter below the age of 16 or 18 years.

Haines, Mayo Clinic, states that cessation of iodine in cases of exophthalmic goiter will be followed, usually within four to six days, by return of the characteristic symptoms.

We think prospective operative cases of exophthalmic goiter can be better prepared for surgery by the operating surgeon, as there is much to be gained by him in his estimate of the surgical risk after he has had some considerable contact with the patient over at least the preparation period of time. He cannot make a proper estimate of the surgical risk in one or two interviews only.

We are of the opinion that, among the rank and file of the profession, too small doses of iodine are given ordinarily; it will require ten minims of Lugol's solution three times a day to hold the symptoms in check, and during a crisis, or for intensive preparation, fifty to a hundred minims a day.

B. TREATMENT OF SERIOUS POSTOPERATIVE ABDOMINAL CONDITIONS.

We think in recent years there has been a tendency not to take advantage of all the accessories in the postoperative treatment of serious abdominal surgical cases. We have drifted more or less into the routine of doing the operation, putting the case on a standardized routine to be carried out by assistants or internes.

Tight dressings following abdominal operations are among the first faults. They restrict breathing, cause discomfort from increased pain and pressure, and certainly have but little to their credit. The duodenal tube for postoperative vomiting and distention is of the greatest benefit:

1. The tube relieves conditions due to gas and regurgitated intestinal fluids. It is a vent pipe.
2. It affects interrupted or continuous lavage of the stomach and, in some cases, of the duodenum.
3. It gives relief from nausea.
4. It makes possible the free drinking of water and thereby relieves that most distressing symptom, thirst.
5. It permits transgastric feeding.
6. It relieves toxemia.
7. It is a port of entry for all kinds of medication.
8. It improves the feelings of all patients; they often beg for its return after they have once experienced the relief afforded by its use.

Murphy drip and large quantities of saline subcutaneously, 1000 c.c. every six hours. In peritonitis, 1000 c.c. hypertonic salt solution 3 per cent every eight hours for three doses. Massive hot packs, as advocated by the late Dr. Ochsner, kept hot by electric pads. When the pulse is inclined to be rapid, we have derived benefit from twenty minims of some good hypodermic preparation of digitalis, often combined with thirtieth or fortieth of strychinine to increase tone of intestinal muscles. Where there is prolonged inability to take food, 1000 c.c. of 2 per cent glucose in normal saline intravenously may be used instead of the normal salt solution.

There is only one objection to the intravenous glucose, that is, occasionally, in spite of all precautions, there will be chill and fever reactions.

C. OSTEO-ARTHRITIS.

In this brief discussion of osteo-arthritis, we are referring particularly to the "creaking knees," characterized by a moderate degree of soreness, giving way, temporary locking, swelling and pain after exercise. These symptoms usually are of long standing. They may, however, occur in young ath-

letes who have had injury to their knees. We operated upon one case with loose bodies and erosions, at the age of 19, following football trauma.

This group of cases outside of the orthopedic hospitals usually pass from doctor to doctor without receiving much benefit and they are excluded from taking part in the more strenuous affairs of life. X-rays often show lipping of the articular margins and very often loose bodies. After these attacks of giving way and temporary locking, there is fluid for some weeks.

We have been neglecting these cases because of the long fear of opening knee joints, a feeling of hopelessness in finding all the loose bodies, and a lack of knowledge as to the relief that may be given aside from their removal; that is, by removing broken cartilages and hypertrophic synovial fluid often containing chondromas. A great many of these cases are benefited and are able to carry on a practically normal existence even after the articular cartilages have been nearly destroyed by removal of the vegetations in the knee joint and loose or unloose bodies.

The above can be accomplished only by very liberal exposure of the entire knee joint, which is admirably accomplished by the incision of Fisher. This incsiion begins above and over the center of the quadraceps tendon, three to four inches above the patella, comes down until the upper border of the patella is reached, then around to the inner side and back to the center of the patellar tendon, and on to the attachment of the tendon to the tibia. The skin and superficial fat are reflected back each way, the outer flap to the outer border of the patella. A straight incision is then made into the fascia, following exactly the same line as the skin incision except that it goes directly over the patella. The fascia is reflected well to the inner side. The synovial sac is then opened along the inner side of the patella and split the entire length of the other incisions. The tendon at the upper border of the patella is seized by heavy vulsella forceps and the patella is completely displaced to the outer side of the joint. The joint is then wide open. For a more complete inspection the leg is then flexed past right angles, exposing the whole upper end of the tibia. The upper compartments of the synovial sac are readily accessible and any loose bodies can be milked into the joint. After the loose or attached bodies are all accounted for, any synovial villi hanging into the joint cut off, the joint restored as nearly as possible, to normal, the wound is closed in three layers: the synovial sac with No. 0 ten-day chromic; the fascial layer with No. 1 chromic; and the skin preferably with clips. The leg is then put on a posterior molded plaster splint with moderate flexion. The leg is elevated in bed. The splints are usually removed in about ten days and passive motion is begun.

D. COMPRESSION FRACTURES OF THE VERTEBRA.

In recent years there has been grat forward movement in the recognition and treatment of compressed fractures of the vertebra. We have learned that they may be caused by trivial accidents and often in the beginning have comparatively trivial symptoms. We purposely say "in the beginning" because, if they are not recognized, the disability progresses for months and months. As these fractures are produced by hyperflexion of the spine, heavy objects falling on the head, or by stiff-legged falls, a carefully taken history of the accident will often suggest an x-ray of the spine. Too often, even yet, many of the cases are going from the third to the sixth week, when it is found that the patient's symptoms are gradually worse, and when an x-ray is taken and a compression fracture is found. It should be absolutely routine in any spinal picture, for trauma or otherwise, that a two-way picture be taken.

After the fracture is diagnosed, the patient should be treated on his back on a convex bed for at least three months, and wear a well-fitted weight-supporting and hyperextension brace for at least a year. Cancellous bone does not heal and calcify as quickly as the harder bones, hence, the deformity is inclined to increase under the body weight. Not only is the body weight taken off of the fracture, but extension is accomplished on a convex bed. This may be easily constructed by simply putting comforts and blankets under the center part of the mattress.

This subject is of special interest to industrial and railroad surgeons, as the time has arrived when we cannot defend ambulatory treatment of compression fractures of the vertebra.

We refer to the following six cases reported by us under the title of "The Unrecognized Compression Fracture of the Vertebra," in 1925, as illustrative of the late recognition these cases were getting at that time.

Case No. 1.—G. D., a school girl, aged 13 years, went down a slide on the school grounds, three and a half months ago, at 9 a. m. She hurt her back and had to lie down for five minutes, but went on a hike at noon. Moderate pain continued, but it was two months before a physician was consulted. The important details in this history are that her buttocks hit the ground, and the nape of her neck, still being on the end of the slide, forcibly flexed her spine. Three months after the injury she had a deformity in the lower dorsal region, the pain was growing

progressively worse, and she was hardly able to be around without support. The diagnosis was a compression fracture of the body of the eighth dorsal vertebra.

Case No. 2.—A. E. S., male, aged 40 years, dived off a springboard into a pool and struck the bottom with the top of his head. This caused some pain in his back, but he finished his swim. It was three months later when he applied for medical attention, at which time he had a spinal deformity and progressively increasing pain, until he was unable to follow his trade as a car foreman. A crush fracture of the body of the eleventh dorsal vertebra was diagnosed.

Case No. 3.—J. D. J., a male, aged 30 years, fell off a motor-truck and "wrenched" his back. He had considerable pain at the time, but went about his duties for four months, at the end of which time the pain was becoming progressively worse, and he had an almost complete disability as a telephone lineman. He could give no exact details as to how he fell. He had a fracture of the body of the third lumbar vertebra.

Case No. 4.—W. A. P., a male, aged 50 years, fell off a carpenter's platform, six feet high, alighting across a saw-horse. He was unable to get up and walk without help, but had no paralysis. He was taken to the hospital and remained there for two weeks, when he had sufficient recovery to be up and about. He continued under the care of physicians, and was not considered seriously injured, as the usual anteroposterior x-ray pictures showed no bony injury. He was finally looked upon as a malingerer. Lateral pictures showed a marked compression fracture of the body of the first lumbar vertebra.

Case No. 5.—W. L. S., a male, aged 48 years, was a brakeman. A log rolled off a flatcar, throwing him backwards, his buttocks striking the ground about two feet from a tree. The upper part of his spine hit the tree, causing hyperflexion. He had some fractured ribs and an injury to his leg. Three months later he continued to complain of pain in his back, and it was discovered that some deformity had developed, and that he had a compression fracture of thebody of the first lumbar vertebra.

Case No. 6.—W. T. E., a male, aged 46 years, a switchman, fell under a car and was rolled in hyperflexion, with his head against the ground. Anteroposterior x-rays were negative. Three months afterward, because he still complained of pain in his neck, x-ray pictures were very carefully made. These showed a fracture of the odontoid process of the axis, from excessive flexion of the neck.

DISCUSSION

DR. P. G. CORNISH, Jr., Albuquerque, N.M.: Dr. Brown, in speaking of compression fractures of the vertebra, reminds me of a case of mine—a man with complete ankylosis of the spine due to arthritis. This man was struck on the back by a rock. He came to the hospital with only a few abdominal pains, no other symptoms. These pains left within 24 hours and x-ray pictures taken at that time showed an old ankylosis of the spine; that a compression fracture of the body of the first lumbar vertebra had been missed, due to the fact, no doubt, that there had been so many arthritic changes. That was in December; in May, he was able to get around and about. He claimed he had paralysis of the bowels, which was chronic constipation. Our x-ray pictures at that time showed gradually increasing atrophy in the body of that vertebra, in spite of the complete ankylosis of the spine. This was quite a lesson to us to be extremely careful in the interpretation of x-ray pictures.

DR R. H. FINNEY, Pueblo, Colo.: Relative to exophthalmic goiter, I have seen results of five cases operated under sodium amytal, which were very gratifying. This was just a preliminary experiment. Fifteen grains were given intravenously previous to operation, the patient sleeping from 12 to 24 hours after operation, with little excitement and apparently good results.

DR. W. L. BROWN (closing): As time is very short, I want to say just a few words. The time has come when the orthopedic men have entered into the field of fracture; we general practitioners hesitate to tell a man he will have to stay in bed three or four or five months, though this is often necessary and should be done.

In regard to the use of sodium amytal in exophthalmic goiter, I remember that in many cases where it was used, it was often quite difficult to get up the mucus. That was our experience last year at Taos during the annual meeting of this Society. Right following that experience I went over to the A. M. A. meeting at Seattle; at one of the hospitals they invited me to se a case operated on with amytal and they had the same difficulty.

SIGNIFICANCE AND TREATMENT OF PLEURISY WITH EFFUSION IN PULMONARY TUBERCULOSIS

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(Read before the Staff of the U. S. Marine Hospita, Fort Stanton, N. M., and published by permission of the Surgeon General, U.S.P.H.S.)

ETIOLOGY

The factors causing a sero-fibrinous pleurisy are in most instances the same as those causing a dry pleurisy. It is thought that over ninety per cent of the cases of pleurisy with effusion are of tuberculous origin. Sometimes pleural effusions are associated with other diseases such as pneumonia, acute endocarditis, and septicemia, as well as with carcinoma, especially of the lung, and other acute and chronic diseases. As an example of this, one of our patients here gives a history of an effusion following scarlet fever, and ten to twelve years later he developed symptoms of pulmonary tuberculosis.

Some cases of primary pleurisy come on after an exposure to cold or dampness or after an overstraining of the body forces. A careful history will often show that the patient has been overworking or has been otherwise neglectful of the welfare of his body for several months or years. Perhaps he has not had the proper diet or sleep, or he has undertaken a long exhausting journey, or has been considerably exposed to inclement weather. A good reason will usually be found for his breakdown. Sometimes an injury to the chest is the apparent direct cause which generally acts as a contributory factor in localizing the tuberculous process to the traumatized pleura. Although a tuberculous pleurisy may be the primary manifestation of the disease, it is commonly secondary to a focus in some other part of the body. This primary focus will usually be found in one of the pulmonary apices; but the infection may reach the pleura from the

periphery of the lungs, the hilus, or a tracheobronchial gland through the lymph channels. In fact, the pleura may become infected through the blood stream from a focus in any part of the body. When a serous effusion complicates a pneumonia, it is not always certain that it was caused by the pneumonic inflammation. Some of the patients at Fort Stanton give a history of having a pleural effusion following pneumonia and of breaking down with pulmonary tuberculosis several years later. In some of these, it is possible that the tuberculous infection was present from the start and was the responsible factor for the effusion, instead of the pneumonia. A serous effusion should always be regarded with suspicion, even when associated with other diseases and a tuberculous focus can not be found. Pleural effusions are more common in men than women. They can occur at any age, but are most prevalent in young adults, as is pulmonary tuberculosis.

SYMPTOMS

Inflammation of the pleura is most frequently located in the apex of the lung. Pain is more commonly associated with pleurisy in the lower part of the pleura, along the anterior axillary line just below the nipple, as this is the site of the greatest lung excursion during respiration. The pain may be very piercing and require the strapping of the chest wall to relieve it. The reason that pain is more rarely experienced at the apices is that the movement of the lung there is minimal.

An effusion is usually preceded by pleurisy. The pain is usually lessened after the fluid forms, as the two pleural surfaces are then separated. The patient may note at this time, or before, some or all of those symptoms so often associated with tuberculosis; as, loss of weight, nervousness and irritability, easy fatigue, slight rise in temperature and pulse.

When the fluid forms there will be an elevation of the temperature. If the effusion is small in amount, the patient will probably not show any marked symptoms. On the side corresponding to the pleurisy he may note a sense of pressure, often a dull pain in the lower part of the chest. The temperature may not be higher than 100 or 101 F., but may last for several weeks. The patient may not be particularly annoyed by the presence of the fluid. Provided he is kept in bed until some time after the temperature has declined to normal, the effusion is generally gradually absobred.

If the amount of the fluid is large, there will be a sense of pressure and tightness in the chest and often a shortness of breath. There is often a dull pain referred to the upper abdomen on the side corresponding to the effusion. The patient will find that he can breathe more comfortably when lying on the affected side, leaving the uninvolved lung free for motion. The temperature may be elevated to 103 or 104 F., but usually subsides after thirty-six to forty-eight hours. In early tuberculosis there is often some nausea and it may be aggravated at this time, due to toxemia. There may or may not be a cough.

DIAGNOSIS

The diagnosis of a pleural effusion will be more easily made when the patient has been seen from the beginning, as close clinical observation is of great assistance. When the patient consults the physician after the acute stage has passed, it may be difficult to make a diagnosis from the physical examination alone, especially if the quantity of fluid is small. It is not always easy to determine between a pleural effusion and a thickened pleura. The use of an exploratory needle may be necessary. It is under these conditions that an x-ray is very valuable and often indispensable. The roentgenogram will help to differentiate between a thickened pleura and an effusion in most cases, even if the effusion is slight. The shadow cast by effusion, if there are no adhesions, is typical. It is a dense homogeneous shadow obliterating the costophrenic sinus and filling the lower part of the lung field. This shadow tapers and becomes less dense as it extends higher in the lateral or axillary portion of the lung field. The balance of the lung is hazy in comparison with its fellow, on account of compression and perhaps also due to a thin layer of fluid covering it. A horizontal fluid level is not seen except in effusions complicating pneumothoraces. No doubt the roentgenologic examination will save many patients from the diagnostic needle puncture.

The exudate should be examined microscopically and an animal inoculation done, especially in those cases where a diagnosis of tuberculosis has not been made. Tubercle bacilli are only rarely found microscopically. A negative examination of the exudate does not exclude tuberculosis as a cause of the pleurisy. Guinea pig inoculations are more often negative than positive and here, too, a negative experiment does not rule out the possibility of tuberculosis.

TREATMENT

In effusions of unknown origin, whenever there is the least symptom pointing to tuberculosis, the patient should be treated as tuberculous, even if no pulmonary disease can

be found upon expert physical and roentgenologic exploration. These patients should be kept under close observation and periodically examined for pulmonary involvement. The physician will not always be able to find evidence of pulmonary tuberculosis, but if the patient complains of nervousness, fatigue, loss of weight or appetite, or other symptoms of early phthisis, he should be treated as if he had the disease. When the effusion follows some other disease, the patient should be carefully watched for signs or symptoms of tuberculosis for several years.

There is a difference of opinion among phthisiologisists as to the advisability of aspiration in the treatment of pleural effusions. If the effusion is small and produces few symptoms, the withdrawal of the fluid is certainly not indicated. Under these circumstances, the patient should be kept in bed for some weeks after he is free from fever, and it will be noted that the fluid will be absorbed in the great majority of cases.

When the amount of fluid is large and the patient is uneasy and dyspneic, it is best to aspirate. Even in these cases the fluid will at times disappear of its own accord if the patient is left alone. But to render the patient more comfortable and also to hasten absorption, some of the fluid should be withdrawn. In some, the fluid will reaccumulate necessitating frequent paracenteses, but in a good many the remaining fluid will gradually disappear in from six to eight weeks. No set rule can be laid down for aspirations; each patient should be treated individually.

The best time to aspirate is after the acute stage has passed. When aspiration must be repeated, it is best to wait for an interval of a week or two. Sometimes the temperature is increased for a few days after the tapping.

The amount of fluid to be withdrawn depends upon how much is present in the chest at that time. Usually from 500 to 1000 c.c. is the quantity taken out at one time and this should rarely exceed 1000 c.c. Violent coughing may be induced when too large a quantity is removed at one sitting. If the condition is of short duration, a greater amount can be aspirated than if it is of long standing. In these chronic cases the lung will not so readily respond to its former position.

When there is definite pulmonary disease, the healing effect of the compression of the lung by the fluid must be considered. This is especially true in acute miliary tuberculosis. In these cases, the pysician should not be too eager to remove the fluid.

At the U. S. Marine Hospital, Fort Stanton, N. M., it is the custom with pleural effusions complicating a pulmonary tuberculosis, to induce a pneumothorax to replace the fluid that has been aspirated. In cases where there is no contraindication, the artificial pneumothorax treatment is continued. Thus the lung is kept at the rest indicated by nature when it caused the fluid to form. It has been noticed, in some instances, that the fluid does not reaccumulate after the substitution of air. In the others, the presence of the serous fluid is not more harmful than the usual effusions complicating pneumothorax therapy. It is best to make the first induced pneumothorax the final step of the aspiration operation, as it is possible for adhesions to form if it is postponed to a subsequent date.

PROGNOSIS

Pulmonary tuberculosis beginning with a pleurisy, with or without an effusion, seems to do better than other types of the disease. This is especially true of those developing their pulmonary pathology more or less shortly after the primary pleurisy. It may not apply in those where there is an interval of several years between the two conditions. Fishberg[1] states: "Acute progressive phthisis following primary pleurisy is extremely rare, excepting in acute miliary tuberculosis, or in acute pneumonic phthisis which, in rare instances, is accompanied, or masked, by a pleural effusion." Bonney[2] says: "The type of pulmonary tuberculosis characterized by initial pleural involvement is generally conceded to be one of the most benign forms of the disease."

During the last few months, two hundred patients have been interviewed at the U. S. Marine Hospital, Fort Stanton, N. M., and their histories gone over, as to whether or not they have had a pleural effusion. This group of patients has been under treatment for a period of from three months to several years, the longest stay being nineteen years for one patient. An attempt was made in every case to determine if the pleural disease was primary or secondary to the pulmonary tuberculosis. A history of pleural effusion was obtained in twenty-nine cases (14.5 per cent). The correct percentage would probably be higher, as small effusions may occasionally pass unnoticed. The roentgenograms of the chests of all these patients have been carefully studied. Every picture taken in every case was examined especially for the presence of an effusion and for the type and extent of the pulmonary tuberculosis. It is a rule in this institution to have chest films made at intervals of approximately six months, on each patient.

Accurate records of the side affected could be ascertained in only twenty-five of

the twenty-nine cases. It was on the right side in ten and on the left in thirteen, and bilateral in two, but not on both sides at the same time.

Nineteen of the twenty-nine cases were aspirated. This was repeated several times in mose of them. In many the fluid was finally absorbed or did not reaccumulate following paracentesis. One patient had eleven aspirations over a period of several years and still has a pleural effusion at the present time. It is not causing him any discomfort and he is able to work fairly hard and is considered to be in fairly good health. In six who have never been aspirated, the fluid disappeared in four; the other two are of recent occurrence.

It was difficult to state exactly, in some cases, whether the effusion or the lung involvement was primary. With the best history obtainable it was thought that the pleural effusion occurred first in seventeen of the twenty-nine cases.

From the standpoint of the roentgenologist it was noted that in patients with pleural effusions, and especially those in whom this was an initial manifestation, the pulmonary lesions were more consistently localized to the apices than in a similar group of other patients. The examination of a series of chest films of the same individuals gave the impression that the pulmonary pathology of patients with effusions progressed more favorably and improvement was more rapid. This is particularly marked in those who cooperate in their treatment. The explanation in part may be that the disease is detected earlier in this type of case. Still it seems that some immunity may also be derived from the pleural effusion.

In the review of the films of the patients giving a history of pleural effusion, it was noticed that many of them showed evidence of an interlobar pleurisy on the same side as that of the fluid. This may be due to the deposit of an exudate in the interlobar space.

To test the prognosis further, the erythrocyte sedimentation records of all the patients were examined. This test is done on practically all patients in this hospital and it is repeated at frequent intervals. It has been found reliable in indicating the activity of the tuberculous lesions. It is realized that, as a measure of tissue destruction, it will be influenced by other diseases besides tuberculosis; but in patients known to have this disease, it can be considered of great value in estimating their prognosis.

According to the Linzenmeier technic, most healthy individuals will show a sedimentation time of over six hours. For clinical purposes a reading above four and one half hours for men and three and one half hours for women would indicate that little or no tuberculous activity was present in the body.

All of the patients in this study are adult males. Sixteen of the seventeen with primary pleural effusions, mentioned above, have had this test. Thirteen of these sixteen (81.29 per cent) showed a sedimentation time of more than four hours. In nine of them it was over six hours. The remaining three (18.7 per cent) had tests of two hours or less.

Since July, 1929, 266 patients had their erythrocyte sedimentation time recorded once or oftener. Of these, 40 per cent were over four hours: 18 per cent between two and four hours; and 42 per cent less than two hours. It will be noted that the percentage above four hours is less than half of that of the primary effusion group. By going through the files of the erythrocyte sedimentation tests, three groups of sixteen patients each were picked out at random. In these the percentages of those having sedimentations of four hours or better were 25 per cent and 31.2 per cent respectively. None are found to compare favorably with the primary pleurisy group.

In this study it was found that pleural effusions seem to be associated with early types of pulmonary tuberculosis. One of the reasons for this may be that in the chronic far-advanced cases the two pleural surfaces are liable to be adherent. When it does occur in the chronic case it appears to be of no material help in the great majority of the cases and may even prove harmful. That effusions do occur in chronic terminal cases is evident from the necropsy statistics of this hospital. Among the last eighty autopsies, there were six cases showing pleural effusions (7.5 per cent). One of these was bilateral.

CONCLUSIONS

(1) Pulmonary tuberculosis preceded by pleurisy or a pleural effusion seems to run a more benign course than the ordinary case.

(2) Small effusions are usually absorbed and should not be aspirated.

(3) Large effusions producing distress should be aspirated.

(4) Aspiration in these cases should be immediately followed by pneumothorax therapy whenever there is demonstrable pulmonary pathology or a positive sputum, and the involvement of the contralateral lung is not prohibitive.

REFERENCES

(1) Fishberg: Pulmonary Tuberculosis, 1922, p.516.
(2) Bonney: Pulmonary Tuberculosis and Its Complications, 1908, p. 351.

METHODS OF REST FOR THE WORSE LUNG—POSTURAL REST AND ARTIFICIAL PNEUMOTHORAX

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(Read before the Staff Meeting of the Good Samaritan Hospital, Phoenix, Oct. 27, 1930).

Tuberculosis, attacking the human lungs, follows a fairly definite course. It begins on one side; sooner or later the opposite lung is involved, more or less than the first. But at some time in the course of the chronic case there is usually a period when the active disease is nearly all in one lung, and it may remain so for a long time.

Rest is the treatment for tuberculosis. When the disease resides mostly in one lung, there is obvious advantage in affording intensive rest to that lung. The method of so doing has to be decided after careful study of the individual. There are no set rules to make such a decision easy, but certain broad principles apply to it.

The safest, the slowest and the least radical way of favoring one lung is by postural rest. This procedure, introduced by Webb and Forster, requires that the patient be in bed. A small firm pillow, reaching from axilla to rib margin, is placed under the worse side, and the patient lies on it, in the lateral posture. The pillow splints the ribs and decreases their motion. This postural rest on the worse side is gradually increased from a few hours to twenty hours a day. The heart and mediastinum are displaced to the affected side, and if that lung is in condition to contract they remain so displaced. The diaphragm on that side has at first a wide excursion, but as the lung contracts the motion of this leaf of the diaphragm becomes limited. In a case suitable to this treatment, a symptomatic improvement is seen in two weeks. If, after faithful trial for two months, no benefit is observed from postural rest, the method should be abandoned.

Supplemental to postural rest is the use of the shotbag. This bag, shaped to the chest, is applied over the upper lobe while the patient lies on his back. The weight of shot, two pounds at first, is gradually increased. The shotbag may be used to relieve the patient for an hour or two from his lateral posture; or it may be employed alone, in apical disease of one or of both lungs, with considerable success. It teaches the patient to breathe with the diaphragm.

Postural rest is simple, safe, and inexpensive, and it is helpful in teaching the patient to relax and to really rest. It can be employed even in the presence of some activity in the better lung, and it is often a useful mode of preparation for collapse therapy. In suitable cases it accomplishes the healing of both lungs, with the advantage that the other mechanical aids are left in reserve for a possible relapse. But postural rest is slow; it does not meet the need in emergency. The patient chosen for posture must have resistance to his disease and not too much damage in the worse lung—say no cavity more than an inch in diameter. He must also have patience, persistence, and money enough to live on. Within these limitations postural rest is a definitely useful measure, particularly in doubtful cases pending decision as to more radical treatment.

The most popular and probably the best mechanical treatment for unilateral tuberculosis is artificial pneumothorax. By this operation, unless adhesions prevent, the worse lung may be put at rest to the extent required for healing. The procedure is not difficult, the danger to life is slight, the degree of collapse can be observed and controlled at will. Provided only that the worse lung is not too much bound by adhesions, and that the other lung is sound enough to carry on, pneumothorax is applicable to the disease in all its stages. It is the operation of choice; massive disease, open cavities, progression in the worse lung, and hemorrhage from that lung, add force to the indication for pneumothorax. So does tuberculous disease of the glottis. If spontaneous pneumothorax occurs on the worse side (and if the other side is in fair condition it may be assumed that the worse lung was ruptured) it should be continued by inflations. Similarly, an aspirated effusion should be replaced by air. When postural rest fails to heal the worse side, pneumothorax should be considered. When the patient with one-sided disease must work or starve, his chances are better with pneumothorax.

But pneumothorax cannot always be used for the relief of one-sided tuberculosis. Even with most of the trouble in one lung, the operation should not be done if the better lung shows evidence of an acute process, scattered areas of caseous disease. The chest film is helpful and necessary in revealing the state of the better lung, but it cannot be trusted to predict whether the worse one will collapse; that can be determined only by trial. A lung opaque to the ray may collapse quickly with a high negative intrapleural pressure, while one that looked clear at the edges may admit no air, or its cavities may be held open by adhesions. These are the cases that call for surgical intervention. They are entitled, I think, to a trial of pneumothorax, first.

With collapse accomplished, the disease in the better lung may advance, demanding cessation of the pneumothorax, and it may be harder to get rid of than it was to induce it. Also there is the ancient bugaboo of effusion into the pneumothorax. This occurs often, but it seldom does harm if it is let alone. Everything considered, artificial pneumothorax remains the procedure of choice for properly selected cases of clinically unilateral tuberculosis.

DISCUSSION

DR. S. I. BLOOMHARDT: I am happy both Dr. Phillips and Dr. Holmes stressed the fact that there has been a constantly increasing tendency to prescribe longer and more complete rest in the treatment of pulmonary tuberculosis and rest has gradually come to be regarded as the most important single measure in the arrest of the disease. The favorable results thus obtained, I believe, have encouraged the wider application of operative procedures which afford more functional rest than can be obtained by bed rest alone. Thus such measures, I am sure, are designed to supplement, not obviate bed rest when this alone has proved ineffectual in selected cases. So often after a pneumothorax or exairesis the patient is permitted by the doctor to forget his lesson of postural bed rest.

In the data showing us the value of pneumothorax we are especially indebted to Saugman of Scandinavia and Matson with his associates in this country. From their data it was first possible to compare the results in large series of cases treated by this method with the result in cases not so treated but otherwise similar, and to appreciate there is a distinct advantage to be gained by its use. Of 1,004 cases of pneumothorax in Matson's most recent series, from 1911 to 1926, 32 per cent were clinically well, 20 per cent arrested, 16 per cent unimproved and 32 per cent died, while in cases selected for pneumothorax when pleuritic adhesions prevented the introduction of gas, only 7 per cent made a clinical recovery as a result of sanitarium care alone, and 55 per cent are dead. Nearly all were severe forms of tuberculosis and were subjected to pneumothorax after failure of other methds.

Other statistics of 570 cases of fibrocaseous tuberculosis in Naveau's study of Rist's cases: 31 per cent were clinically cured, 17.5 per cent improved, 17.5 per cent stationary and 34 per cent unimproved or dead.

Matson and Bisaillon present their results for comparison and find that, of 423 cases of fibrocaseous and fibrocaseous cavernous tuberculosis, 32 per cent were clinically cured, 20 per cent arrested, 16 per cent unimproved and 32 per cent died.

They tell us in their experience pneumothorax is limited with application, as in more than half the cases in which it is indicated a satisfactory collapse, upon which a successful treatment largely depends, cannot be obtained because of adhesions. Of cases in which a satisfactory collapse was established, 48 per cent are clinically well, 20 per cent arrested, 11 per cent slightly improved and 21 per cent dead; whereas, in cases with only partial collapse, 13 per cent are clinically well, 13 per cent arrested, 15 per cent slightly improved or unimproved, and 50 per cent dead.

Embolism is the most immmediate danger of neumothorax. In their 12,000 inflations, the "gas embolism shock" occurred 19 times but only 2 cases were fatal. Saigman had only 2 embolic deaths in 11,500 inflations. Tuberculous empyema occurred in 12 per cent of 480 collapsed cases, but was not a serious complication unless drainage was established.

Intrapleural pneumolysis we would like to have had on the program tonight, as well as the latest status on extrapleural thoracoplasty, but time did not permit and at a future meeting, perhaps, these two phases of chest surgery can be talked about.

Matson's report of 90 cases of phrenicotomy finds the most striking benefit in patients with fibro-caseous cavernous tuberculosis, stationary or slowly progressive, and in good general condition with a good contra-lateral lung. The results in the exudate types were unsatisfactory. He claims it should precede every thoracoplasty. Occasionally so much benefit follws this relatively simple procedure that the more radical measures are unnecessary. It is relatively little value as an independent procedure; it is not wholly without danger from accidents in connection with the surgical procedure; it is a question whether paralysis of the diaphragm may not eventually lead to more or less serious disturbances. Unilateral phrenic paralysis produced by operation displaces thoracic and abdominal organs, as in the anomaly known as eventration of the diaphragm. A majority of patients with eventration suffer from more or less troublesome symptoms due to the displacement of the viscera and it is desirable to know whether or not similar disturbances are to be expected after operative paralysis of the diaphragm.

RELAPSING FEVER
(Febris Recurrens, Ruckfallfieber, Spirillum Fever, Tick Fever).

KIMBALL BANNISTER, M. D.,
Phoenix, Ariz.

(Abstract of a paper presented before the Maricopa County Medical Society.)

DEFINITION

Relapsing fever is an acute infectious disease characterized by recurrent attacks of fever which are usually separated by afebrile and otherwise symptomless intervals. It is caused by several varieties of spirochetes which are transmitted to man from ticks and lice.

HISTORY

1741—Rutty in Dublin observed the disease associated with typhus fever. 1857—Livingston in South Africa reported it following bite of tick. 1868—Obermeyer reported large spirillum present in blood. 1872—Spirillum confirmed in a German epidemic. 1904—African tick fever proved due to spirochete.

INCIDENCE

I.—Chief centers of distribution, for **louse-borne** type: European Russia; Poland; Balkan States. Occasionally epidemics. 12,000 cases in Serbia during World War.

Mild epidemics occasionally occur in: China, India, North and West Africa. 600 cases in Gold Colony of West Africa in 1922 and 1923.

During 17th and 18th centuries epidemics occurred in: Ireland, Scotland, Aus-

tria-Hungary, Germany, Russia. (1869 imported from Ireland to New York and Philadelphia but short-lived.)

II. Chief center of distrbuton for **tick-borne type:** German East Africa. Spreads along main caravan routes, especially in: Uganda, Congo Free States, British Central Africa, Portugese West Africa.
Endemic in: Palestine, Persia, parts of Central and South America.
Sporadic cases uccur in: Mexico, Southern California. Only one case reported to State Board of Health in Arizona.

ETIOLOGY

Due to spirochete of the genus spironema. (Differently named in different parts of the world but morphologically showing no constant differences.)
Spironema recurrentis (Lebert, 1874). Length 18-23 micra. Breadth about 0.3 micron. 3-6 turns.
Incubation period (7 to 12 days). About one week for it to appear in blood after gaining entrance. Disappear and reappear at weekly intervals several times. Mode of transmission: By crushing louse on skin spironema enters through abrasions. By secretion from tick. By carriers (flies, mosquitoes, or bedbugs) from patients.

MORBID ANATOMY

No characteristic postmortem findings. May find spironema in spleen and bone-marrow.

DIAGNOSIS

Find causative organism in blood **during paroxysm.** (Dark field or Wright's stain).

Differential:

Malaria, Malta Fever, Typhus Fever, Dengue, Smallpox, Trench Fever, Infectious Jaundice, Yellow Fever, Typhoid Fever, Influenza, Pneumonia.

PROGNOSIS

Self-limiting (Succeeding relapses less severe). Usually not fatal except in weak, poorly nourished individuals. 4 per cent fatality in Europe. As high as 30 to 40 per cent in India and Africa. Partial immunity conferred. (May last only 2-3 months).

TREATMENT

Arsphenamine intravenously. (0.2 to 0.3 gm. is usually sufficient).

REPORT OF CASE

Mr. C. E. M., a white male, 37 years old, previously in good health. On August 8th, after an 18-day vacation spent around Greer, Arizona, reported to his physician with a temperature of 103°, stating that he was "sicker than he had ever been in his life," aching from head to foot, but physical examination otherwise practically negative. His condition was attributed to a "cold," but failed to improve, so that 3 days later, August 11th, his temperature had mounted to 105.5°, but still with negative chest and physical findings. To account for the high temperature at this stage a consultant favored an intestinal infection. Following medication by mouth along with cold soda enema, tepid sponges and alcohol rubs, his temperature dropped to around 97° where it remained for several days.

Following this, he had repeated attacks of hyperpyrexia (106° at one time) with ensuing subnormal temperature for several days followed by a period when he felt well and able to continue with his business. His attacks of hyperpyrexia associated with general malaise were uncanny in their regularity, usually starting the temperature rise early in the morning, reaching the peak (104 to 106 degrees) that same evening with the consequent "breaking" early the next morning. Altogether, the patient had seven attacks with relapses occurring at intervals of two to thirteen days (August 8, 21, and 28, September 5, 12, 22 and 24).

The blood Wassermann, urinalysis, Widal reactions for typhoid and paratyphoid "A" and "B", the agglutination tests for Malta fever, and the search for malarial parasites, were all negative. On September 13 the white blood count was 13,700, with 69 per cent polynuclears, 17 per cent lymphocytes, 13 per cent monocytes and 1 per cent eosinophiles. The stool examination revealed no parasites, blood, nor tubercle bacilli.

On September 25, during a paroxysm, a blood smear showed the presence of spironema in the blood, following which one intravenous injection of 0.6 neo-arsphenamin has given lasting relief.

URETERAL STONE SIMULATING ACUTE APPENDICITIS

(Case Report)

W. O. SWEEK, M. D., and
G. C. FRENCH, M. D.
Phoenix, Ariz.

(Presented at the Staff Meeting of the Good Samaritan Hospital, Phoenix, Sept. 29, 1930.)

We have all observed cases of acute pyelitis, acute salpingitis, and ureteral stone, with all the cardinal signs and symptoms of acute appendicitis. These cases arouse a great deal of immediate interest in the mind of the diognostician since the treatment is necessarily quite different. However, when a case is referred to a surgeon with a diagnosis, it is often the rule that no further study is made of the case until the patient is under an anesthetic upon the operating table. Such confidence in your internist without a doubt is seldom misplaced if he has available the various laboratory facilities for confirming the clinical diagnosis. However, if such is not the case, it probably is wiser to do some of the simpler tests, at least before any operative procedure is undertaken. For we are sure it is the general observation that, even with the most complete analysis of a case, at times we are unable to be absolutely positive of the diagnosis; otherwise, the term "exploratory laparotomy, would long since have become obsolete.

Cope mentions a case in which all signs and symptoms, both clinical and laboratory, pointed toward ureteral stone. Even x-ray demonstrated such; however, it was found to be a concretion in the vermiform appendix. Many other similar experiences could be mentioned. If there are any ironclad clinical signs or symptoms which will differentiate between ureteral stone and an appendicitis in all cases, we have, in our experience, been unable to find them.

The case to be reported is not an ideal one, since it is of the type so often met with, in which the finances are so limited that a proper study cannot

be made. There should be some solution to this problem if we expect to do scientific medicine and surgery.

The doctor who referred this case to us stated that this case was to be decided between acute appendicitis and ureteral stone.

Mrs. X came into the hospital at 1:30 a. m, with the following history:

Chief Complaint: Sharp, constant pain in right side of abdomen at level of umbilicus, radiating posteriorly upward to right kidney region and down toward right groin. Was constipated. Pain began four days previously with a sudden onset, continued four or five hours. Was given a liquid medicine which relieved pain. Pain was preceded by a frequency of micturition and was associated with nausea and vomiting. Four days later at 1 p. m. she had a recurrence of the pain, frequency of urination, nausea and vomiting. Pain was sharp in character at onset, but suddenly, after several hours, ceased. However, she was sore in right kidney region and over right abdomen at level of umbilicus. She stated that she had had a similar attack six years previously.

Past History: Usual diseases of childhood. No other illness except as mentioned above. Married 15 years; 3 living children. One miscarriage, 10 years ago, not induced. Menses normal. Leukorrhea since first child was born, 12 years ago.

Physical Examination: Reveals a young adult female lying in recumbent position in bed. She is about 5 feet 6 inches tall and poorly nourished. Skin is moist and warm. Respiration 40, shallow and mostly thoracic. Face shows expression of anxiety. Temperature, 99.4. Blood pressure, 118/80.

Tongue: moist, covered with grayish film. Head and neck otherwise negative. Chest: sthenic type; expansion equal on both sides; no breast tumors. Lungs: no abnormal areas of dullness; breath sounds normal; no rales heard. Heart: no enlargement; sounds clear; rate regular and rhythmic; no murmurs heard.

Abdomen: Slightly distended and tympanitic. Slight hyperesthesia of skin to the right of umbilicus. No rigidity. Pain and tenderness elicited upon deep pressure over McBurney's point. Slight tenderness over suprapubic area, much tenderness over right kidney, the lower pole of which was palpable. No other points of tenderness.

Pelvic examination reveals retroversion of uterus and lacerated cervix. No masses nor points of tenderness.

Rectal examination, negative.

Reflexes normal.

White blood count, 15,200; hemoglobin, 80; polymorphonuclears, 84; small mononuclears, 13; transitionals, 3.

Urinalysis (catheterized specimen): yellow in color; acid in reaction; specific gravity, 1025; acetone, negative; albumin, negative; sugar, negative. Blood, 3-5.

X-ray: reveals two crescent-shaped shadows in right kidney region, about ½ cm. by ¼ cm., which resemble kidney stones. In urinary bladder there are several oval shadows the size of large peas which also resemble stones. Due to considerable gas in belly, re-examination was suggested.

Re-examination: Reveals same bodies in region of right kidney, also an oval-shaped body in right bladder region on a level with the plane passing through the lower portion of first segment of the coccyx.

Nonprotein-nitrogen, 30 mg. per 100 c.c. of blood.

P.S.P. test: first hour, 10; second hour, 16; total, 26.

Cystoscopic examination: bladder normal except for edema of right ureteral orifice. No. 6 catheter ascended to right kidney easily. No. 5 catheter, passed along beside it, was arrested 1½ cm. within ureter. Catheter left intact for 18 hours.

Diagnosis: Ureteral calculus.

Patient was in the hospital 7 days. Temperature ranged from 99.2 to 101.2 until afternoon of fourth day, when it came down to normal and remained so.

Urinary output first twenty-four hours, 75 c.c. Intake, 3000 c.c. Output increased daily, but at no time was the relation better than 4530: 2750.

Pain and tenderness of abdomen and right kidney area gradually subsided until she was free from any discomfort upon discharge.

White blood count was 8600; hemoglobin, 85; polys, 83; small monos, 17.

No further examination was made of kidney function since patient felt well and desired to go home without further expense.

DISCUSSION

DR. H. D. KETCHERSIDE: Dr. French is to be commended on the masterly way in which this case has been handled. The number of cases with renal calculi that have had their appendices removed without relief of their symptoms, bear mute testimony to the fact that these cases are not always so carefully handled. The problem with which he was confronted is much more difficult than is commonly realized. We shall have to grant that, if given sufficient time, most urological diagnoses can be worked out with an amazing degree of accuracy, but, in these cases which simulate acute appendicitis, time is precious and delay often disastrous.

The signs upon which we are accustomed to relyl most, are the location and character of the pain and tenderness and the presence of blood in the urine. We are all looking for the old familiar syndrome of renal colic, severe pain coming on suddenly in the back and radiating down the ureter to the testicle, penis, or inner aspect of the thigh; when, as a matter of fact, that syndrome is seen most often in ureteral calculi, particularly where the stone is lodged low in the ureter, and is not often seen in renal calculi. Pain radiating to the back is not uncommon in acute appendicitis, particularly in retrocecal appendices.

In renal calculi the amount of blood in the urine is, as a rule, not large, and may be found only on microscopic examination and if the symptoms resemble those of appendicitis it is very difficult to differentiate the two. The presence of blood in the urine is not at all rare as the result of systemic infection in appendicitis. It can seldom be explained as due to the proximity of the inflamed appendix to the ureter.

The time has passed when one can make a diagnosis of renal or ureteral calculi from the history alone. The pain, hematuria, pyuria and all the other symptoms, which stand out in the individual clinical pictures, are of great value only in pointing to some condition in the urinary tract. The true nature of the condition can be determined only by a complete urological examination.

Plain radiography, supplemented by the use of the opaque catheter, and ureteropyelography, show the presence of a calculus in about 85 per cent of cases. The use of uroselectan will probably increase the percentage, as it makes possible the taking of pyelograms where it is impossible to pass a catheter up the ureter, and we get our best pyelograms where there is an obstruction in the ureter allowing the opaque medium to concentrate above the point of obstruction.

PRE-ECLAMPTIC TOXEMIA
(Case Report)

C. N. PLOUSSARD, M. D.
Phoenix, Ariz.

(Presented at the Staff Meeting of St. Joseph's Hospital, Phoenix, Oct. 13, 1930).

Patient was sent into the hospital July 28, 1930, by Dr. Fournier, with a diagnosis of pre-eclamptic toxemia and approximately seven and a half months' pregnancy. White adult female, age 39 years. Primipara. Has been married six years.

Family History: Father dead, blood poisoning. Mother dead, tuberculosis. No miscarriages.

Complaint: Patient states her feet and ankles began to swell one week ago. Much indigestion and gas on stomach. No vomiting. Soreness in wrists and ankle joints. Small black specks before the eyes at frequent intervals. Pain in the abdomen when lying on the right side. Appetite good. Urination infrequent and scanty, with pain when urinating. Constant headache for the past two days.

Past History: Had an appendectomy ten years ago; adhesions six years ago. Right mammary gland partially removed four years ago for tumor. Menses regular, every 28 days, duration three days, many clots and cramps. Last menses, November 28, 1929.

Physical examination shows an adult white female lying on her back but restless, with eyes open wide. Breath hard to get. Skin is sallow, edematous condition over the whole body, more marked on the feet, hands and eyelids. Neck: carotid pulsations very evident. Chest shows heart sounds increased in intensity. Right breast small due to operation four years ago. Otherwise, chest normal. Abdomen is elarged from below upwards to four finger breadths below the ensiform. Has three old scars in the midline between the symphysis and the umbilicus. External genitalia normal. Edema of vulvae. Extremities normal except for edema.

July 29: Patient's condition did not change much the first day in the hospital. Blood pressure mounted to 172/100. Profuse perspiration.

July 30: Patient had very restless night. Jumps in bed at the slightest noise. Has constant headache. Never slept at all. Has spots before the eyes constantly. Blood pressure had mounted to 196/115 and patient seemed on the verge of convulsion. Eyes were blinking and extremities jerking. Could hardly talk to her because the voice would make her jerk and jump. She was given 20 c.c. of 10 per cent magnesium sulphate into the vein. Aspirin for headache. Urine showed .125 per cent albumin. Blood: Hemoglobin, 80 per cent; lymphocytes, 23 per cent; polys, 77 per cent.

Because of the fulminating type of toxemia and the absolute failure to respond to the usual medication, rapid delivery by cesarean section was thought the best procedure for both mother and child, the patient being on the verge of a convulsion.

Under sodium amytal, 10 grams into the vein, supplemented with ether, a right paramedian incision eight inches long, between symphysis and umbilicus, was made and the abdomen opened. Uterus was brought into the wound. Intestine was packed away from the uterus and uterus incised. Bleeding was more profuse than usual, probably because of the patient's high blood pressure. Amniotic sac was opened and a baby girl weighing 4 pounds and 8 ounces was delivered. There was injected into the uterine muscle 1 c.c. of pituitrin. The uterus was sutured in three layers and the abdomen closed in layers.

Post-operative report:

First day: Returned from surgery in fair condition; temperature, 98; pulse, 84. Patient very restless. Was given one-fourth grain of morphine sulphate. Pituitrin, 1 c.c. Later, pituitrin repeated because of more than usual amount of bleeding. Caffein sodium benzoate was given. Pulse not rapid but weak.

1st night: Temperature went to 101 4/5; pulse, 110. Patient very restless but with no pain. Much indigestion.

2nd day. Still complaining of indigestion and shortness of breath. Began urinating small amounts frequently. Morning temperature, 99.6; pulse, 96. Afternoon temperature, 101.2; pulse, 100. Patient was given 500 c.c. citrated blood into the vein.

2nd night: Temperature, 101; pulse, 100. Quite restless and nauseated. Stomach lavaged. Given glucose and saline into the vein. Complaining of severe headache and gastric distress and unable to get breath.

Edema still marked over entire body with rales in the chest.

3rd day. Patient's condition worse. Temperature, 98; pulse, 94. Unable to get breath. Patient cyanotic. Given oxygen and stimulation. Alpha-lobelin given, and 500 c.c. more citrated blood into vein. Patient became more restless, more cyanotic. Edema of the entire body, uvula, larynx and lungs. Pulse became rapidly weaker. Generalized edema seemed to change little. Patient died.

Blood pressure readings: Fetal heart rate

4/8/30	120/70	154
4/29/30	110/70	157
5/23/30	98/60	163
7/5/30	120/70	169
7/27/30	152/90	171
7/29/30	172/98	
7/30/30	196/115	176

Total amount of urine for three post-operative days, 300 c.c. Death on the third post-operative day, with practically normal temperature and very little distention, probably due directly to kidney destruction and secondarily to pulmonary edema.

Baby girl is doing nicely.

DISCUSSION

DR. F. C. JORDAN: Pre-eclamptic toxemia is not uncommon and may occur in patients who have had the best of pre-natal care. By far the largest number of patients who develop pre-eclamptic toxemia have had inadequate and, in many cases, no pre-natal care. Every pregnant woman should see a competent physician early in her pregnancy. Her physician should take careful history, make accurate pelvic measurements, and make a complete physical examination. She should see her physician at least once every three weeks and her blood pressure should be taken; also her urine examined. Some physicians neglect to take the blood pressure of their patients. I am sure this is a serious mistake for, I believe, in many cases, the blood pressure readings will be altered before there are any abnormal urinary findings. The average blood pressure in pregnant women is between 110/70 and 120/80. If the blood pressure should go up to 130/90, the patient should be closely watched. I have seen one case of eclampsia in which the blood pressure had not been over 132/90. High blood pressures alone are not at all serious, but they bear close watching. High blood pressures with only a trace of albumin are not as serious as

those with larger amounts of albumin. The presence of casts and albumin in the urine and high blood pressure always are of serious importance. These patients should be immediately placed in the hospital and be under the constant care of a good obstetrician. Eclampsia occurs in primipara about ten times as frequently as in multipara; also, eclampsia often recurs in subsequent pregnancies.

How shall we treat pre-eclamptic toxemia? Strogonoff's conservative method, which he introduced in 1897 with such a low mortality, has superseded all other forms of treatment. He gave large doses of morphine hypodermically and chloral by rectum, and the patients were kept abslutely quiet in a dark room. In later years, intravenous administration of magnesium sulphate and glucose solutions have seemed to aid materially.

Plasmapheresis was performed eighteen times in fifteen patients in the Boston Lying-In Hospital. In five eclempsias, prompt recovery followed. In five cases of pre-eclamptic toxemia where other methods, including induction of labor, had failed, reduction of blood pressure and disappearance of albuminuria ensued. It was also tried in five chronic nephritics. These cases showed temporary improvement. The following technic was used:

About 1,000 c.c. of blood was withdrawn (depending upon the size of the patient and blood pressure readings.) The blood was mixed with sufficient citrate solution to prevent clotting. The citrated blood was then centrifuged for 20 minutes and the serum siphoned off. Then 500 c.c. of normal saline solution was added to the corpuscles and thoroughly mixed and centrifuged for 20 minutes. The fluid was again siphoned off and 100 c.c. of normal saline was added to the remaining corpuscles. This was re-injected into the vein. The red count showed very little, if any, diminution. It would seem that venesection followed by a transfusion would do equally as well, provided a suitable donor could be secured.

I think this chart should be critized in that there is only one blood pressure finding recorded. There were notes under the treatment orders that the blood pressure should be taken twice daily but no records were charted. The only recorded pressure, of 105/115, was made on the second day in the hospital. All obstetric patients should have their blood pressures taken and recorded on the day of entrance. No doubt this was taken but it was not recorded.

Summary: Adequate pre-natal care will prevent many of these pre-eclamptic toxemias. If they do occur, the patient should be placed in a well-equipped hospital and given conservative treatment as is indicated in the individual case. Personally, I favor salines for thorough gastro-intestinal evacuation; large amounts of glucose, intravenously; luminal sodium as a sedative. In some cases, there may be such extensive damage to the liver and kidneys that recovery is impossible. Fortunately, these cases are not frequent. If these cases can be well treated in the hospital, the mortality rate should be very low.

DR. F. B. SHARP: I want to add a word of consolation to Dr. Ploussard, as these pre-eclamptic toxemia cases are mighty hard to help. Drainage of the kidney is not going to do these patients much good. I can, perhaps, offer a suggestion or two that would not be a criticism. I notice the question of some ether at the operation; the general anesthesia is noticed to increase substances in the blood stream that are present in eclampsia; however, the fulminating type is often fatal, as it comes up so rapidly. This just teaches us a whole-hearted respect for the pregnant woman and the difficulties we are likely to meet.

DR. PLOUSSARD: This patient was admitted to the hospital late on the 28th, there wasn't much time. We gave her intravenous magnesium sulphate with practically no result. The blood pressures which you see on the chart were on our records at the office and I copied them from there. I found nothing that would come near quieting her. At the sound of a door or a voice outside, she would blink her eyes, and one would think she was on the verge of a convulsion any minute.

DR. J. D. HAMER: There were sixteen cases in the Good Samaritan Hospital during the month of April who came in with pre-eclampsia. We used magnesium sulphate in nearly every one of them. I think the trouble with cases of that sort is that we don't use enough of it, or use it frequently enough. One case came up very quickly and was in a convulsion when she came into the hospital. We did not hesitate to give, every hour, an ampule of 50 per cent and sometimes two ampules of 50 per cent. Not only that, but the beauty of magnesium sulphate is manifested in another way—that it is a good diuretic as well as working on the bowels.

The serious cases, who were given frequent and constant magnesium sulphate, it seems were those who got the better results. There were sixteen cases of convulsions and a number of cases of threatening pre-eclampsia during this one month. We took blood pressures every three or four to six hours, depending upon the severity of the case, and when it had reached what we thought was the maximum, we shot an ampule of magnesium sulphate and an hour later would notice a difference in the blood pressure.

TRAUMATIC TETANUS
(Case Report)

THOMAS W. WOODMAN, M. D.
Phoenix, Ariz.

(Presented at the Staff Meeting of St. Joseph's Hospital, Phoenix, Sept. 15, 1930).

The staff are entirely familiar with the symptoms and progress of a tetanus case, but I have felt that this particular case is worthy of presentation due to the fact that the patient had the antitetanic serum in the usual 1500-unit prophylactic dosage and subsequently developed tetanus. This is rather uncommon and for this reason I feel that we may learn considerable from this one case.

This young man, B. A., entered the hospital on July 27, 1930, at about 9 p. m. He had been in an automobile accident in which his car had overturned into an irrigation ditch. He had a dislocation of the left shoulder with a fracture of the humerus, and a very severe laceration of the right hand, together with compound fracture of the middle and fourth metacarpals. The laceration of the hand extended across the palmar surface and also across the dorsum without severance of the tendons. There was little bleeding and the wound was very dirty.

The dislocated humerus was reduced under gas anesthesia and the hand was cleansed as thoroughly as possible with alcohol, ether and iodine. Interrupted dermal sutures were inserted and through and through drainage established. At that time, within about 1½ hours of the time of the accident, 1500 units of antitetanic serum was given. The hand was then placed in a continuous warm Epsom salt bath.

It is interesting to note that at 4 a. m. of the night of admission this boy's temperature was 103.6. The following day the middle finger of the injured hand appeared quite dark, but it was still hoped that some blood supply might be established. The finger had to be removed, however, on the 31st of July. The temperature promptly subsided to a maximum of 100 and by August 8th the wound was quite clean.

When I saw the boy on the morning of August 8th, he was complaining of some stiffness of the jaw muscles, which was the first symptom complained of. He was promptly given 50,000 units of anti-tetanic serum intramuscularly. By evening his condition seemed to be markedly improved. The following morning there was considerable rigidity with spasm of the diaphragm and laryngeal muscles, and, under ether anesthesia, 50,000 units of antitetanic serum was given intraspinally after withdrawal of some spinal fluid. The temperature rose to 101.8 and, due to the fact that there was very marked rigidity and extreme pain, sodium amytal was given at 7 p. m. Relaxation was immediate. He awakened from the sodium amytal at about 10 but shortly thereafter became cyanotic and died at 12:30 a. m.

There are two outstanding features in the case that are suggestive. The first one is that the rapid rise in temperature such a short time after the accident certainly meant a most virulent type of infection. The second is that the usual prodromal symptoms of tetanus were not complained of.

The incubation period in this case was twelve days, while the usual incubation period in tetanus is from four to ten days, although it may be considerably longer. It is an established fact that, in the cases that do develop tetanus even though they have had prophylactic serum, the incubation period is frequently prolonged. Frequently tetanus is insidious in its onset and the prodromal symptoms are overlooked, yet it is of the utmost importance to make the earliest possible diagnosis, for there is little chance of recovery if the diagnosis is made after the tetano-toxin has combined with the nerve cells.

The prodromal subjective manifestations are a slight feeling of tension or contraction, especially in the region of the wound, slight difficulty or pain on swallowing, cramp-like pain in the muscles, especially near the wound. One important feature is that these cramp like pains will arise particularly when the patient is startled by some noise. The early objective manifestations are twitching of the muscles in the region of the wound, a tendency toward sweating, restlessness, anxious expression of the face, and a slight increase in pulse rate. These symptoms may last for several days before the acute toxic symptoms appear and, generally, progressively increase with the toxemia. The locking of the jaw may occur early or late in the disease.

I am not going to go further into the symptom complex for I feel that all are familiar with the picture. It should be emphasized that the search for the early symptoms listed above is of the utmost importance if we expect to see these cases recover, for the early administration of antitetanic serum in massive doses is our one and only hope.

Sodium amytal given in relaxing doses of 3 to 4 grains is of considerable value.

It has been demonstrated that the immunity following the injection of antitetanic serum lasts about seven days, and that the absorption of tetanus toxin is much more rapid than the absorption of antitoxin; e. g., if the serum and the toxin are injected simultaneously at different points, The toxin which is not neutralized causes mild lo- The toxin which is not neutralized causes milld local tetanus, which is almost always curable.

As undoubtedly occurred in the case I am citing, the immunity was temporary. The spores, inhibited by phagocytes and by the serum, again developed, producing the toxin, which poisoned this boy at a time when no antitoxin was present to neutralize the toxin.

I feel that some of these deaths could be prevented if we would give a second dose of prophylactic serum three to five days after the initial dose, particularly in the cases presenting extremely dirty wounds at the time of admission to the hospital.

SUBPHRENIC ABSCESS
(Case Report)

F. J. MILLOY, M. D.
Phoenix, Arizona

(Presented at the Staff Meeting of St. Joseph's Hospital, Phoenix, Sept. 15, 1930).

Case No. 20262, male, age 41, entered hospital June 28th, last. Could give no history except that he had not been feeling well for four or five days. He stated that he had never been sick in his life before. He had no pain, his appetite was very poor, although he had not vomited before entering the hospital. At the time of entrance, his temperature was 99.6; pulse, 116; respirations, 30. White blood count was 31,950; polynuclears, 90 per cent. The day following admission, patient's temperature was down to 96.6, but that night rose to 104. His blood culture, Malta fever agglutination and Widal were all negative and stools contained no parasites nor occult blood. After the third day in the hospital, temperature ranged between 96.6 and 101, and was below normal more of the time than it was above normal. Pulse ranged from 70 to 120 and respirations from 20 to 40. On July 3rd, the white count was still 31,000 and as yet there were no localizing symptoms. By the 5th of July, the liver was definitely palpable below the right costal margin. It was firm, smooth, and no evidence of pain or tenderness was present. A soft fluctuating mass about the size of half a grapefruit appeared just below the ensiform. This was not tender either. Up to this time the patient had slept well at night, complained of no pain whatever, but was practically unable to eat. The diagnosis at this time was abscess of the liver. The patient was given one-half grain emetine hypodermically. On July 6th, the white count was 25,500; icteric index, 15. X-ray at this time showed stomach normal and evidence of a mass anterior to it. Also, right chest almost filled with fluid with a small amount of air present. On July 7th, white count was 42,000. On July 9th, it was 22,000. At this time the liver border was at the umbilicus. Patient had been receiving emetine every day and seemed to be feeling better and, as the white count had dropped to 22,000, his condition seemed improved. On July 10th, the white count had jumped to 34,000 again, so, on the morning of July 11th, an exploration needle was introduced into the ninth interspace, needle pointing slightly upward. A bloody fluid was withdrawn, all of this fluid was aspirated with the suction. This fluid later coagulated. The needle was brought out and re-introduced, pointing downward, and a cavity was entered and very thick pus withdrawn. Needle was left in position and pus continued to drip through it. An incision

was made immediately below through the chest wall and diaphragm and a large subphrenic abscess was opened and about a quart of pus drained away. Two large garden-hose drainage tubes were introthrough wound near each end. Sutures were tied and wound closed. Drainage continued very profusely following operation. Patient's condition seemed quite satisfactory, took large amount of fluid by mouth. The following day, patient's pulse began to rise and was rather weak and thready and he was in a cold perspiration. He was given 500 c.c. of blood intravenously. His temperature began to rise and was 108 by axilla just before he died at 7 p. m.

The following are the autopsy findings: When attempting to embalm the body and when trocar was passed into the liver area, a large amount of thick pus was encountered which would not come through the aspirating trocar, necessitating an incision into this area for embalming sufficiently well to ship the body. Examination of this area revealed a bulging mass in the epigastrium, which was found to be a large, well-defined abscess cavity with necrosis of the abdominal wall clear up to the skin. Examination of the surface of the liver revealed a liver abscess about the size of the fist with much necrosis and inflammation of the liver tissue. This was just medial to the gallbladder. Examination of the subdiaphragmatic area revealed a large extensive abscess in this area. Examination of the right thorax revealed the hydrothorax containing a large amount of straw-colored fluid. The appendix was bound down by adhesions, showed subacute inflammation which, with the history of the case, was no doubt responsible for his subdiaphragmatic abscess, liver abscess and hydrothorax condition.

DIAGNOSIS

Appendicitis, subacute
Liver Abscess, subdiaphragmatic.

DISCUSSION

DR. R. J. STROUD:—In taking up the discussion of the case in question, the first definite sign of localization was in the fact of a liver swelling—this after Malta fever and typhoid fever had been ruled out. Also an independent swelling was found in a soft fluctuating mass the size of half a grapefruit, below the ensiform.

The fact that these places were not tender and that the patient did not complain of pain was rather deceiving, for it is usual to have pain with pus under pressure, and most certainly it is usual with appendix troubles. I have seen a case of ruptured appendix where all of the pain and tenderness were around the kidney, or at the gallbladder region. Just why this case showed no tenderness or referred pain is hard to tell.

The white count fluctuated but was always high, the signs definitely pointing to a liver abscess and confirmed by exploratory needle; operation to relieve patient of pus. The operation that was done, however, did not alleviate, for there were other foci or abscesses which were still present and which evidently killed because of the added shock of the operation.

It is rather axiomatic, as Cabot says, to look for one cause of a trouble. In this case there probably was one cause but its effect was to make multiple abscesses instead of one. The pathologist did not state whether there was any connection between the liver abscess and the subdiaphragmatic abscess. Very often in these cases there is a connection where the liver abscess has broken through the posterior abdominal cavity and forms this second abscess. It is not usual in a condition of this kind for a patient to recover, as there may be very few symptoms in one of these abscesses below the diaphragm and in the lesser peritoneal cavity, for the space is large and there can be a great quantity of pus present before pressure gives any indication of the enormous amount of pathology which may be in this region.

The causes of subdiaphragmatic abscesses, or abscesses in the lesser peritoneal cavity, are hematogenous infection from infection of the gallbladder, tonsils, sinuses, appendix, pericardium, or other focus. They are also due to the rupture of a liver abscess, rupture of the posterior surface of the gallbladder, rupture through from above of an empyema, rupture of an abscess of the pancreas head or an abscess around and about the kidney. It does not definitely state whether this abscess lies in the lesser peritoneal cavity and suggests that it is just below the abdominal wall up to the skin and it could have been contained in the epigastrium in front of the stomach and between the liver and the costal margin of the left side.

The differentiation between these various abscesses is very difficult. When the pancreas ruptures and pus gathers in the cavity for this reason, it generally follows along toward the left side and the diagnostic sign in this case is a pain over the left kidney, with tenderness going down toward the left thigh, whereas in a rupture of a liver abscess this same pain is very seldom present, as was the case in this instance. In a rupture of the gallbladder posteriorly, great pain from the empyema is first felt and then suddenly there is an alleviation of the symptoms, with a return when the cavity behind fills up. In these cases tenderness has thrown a wall of adhesions to keep the pus from entering the large abdominal cavity. The dome of the liver must have been high, but the amount of fluid in the right chest probably obscured this sign. The lesion evidently causing most of the symptoms was the subdiaphragmatic abscess.

The causes of liver abscess are as follows: Dysentery—this may be a completely latent disease, even if our improved technic often finds the ameba present. Negative stools in this case probably denoted no ameba but could not be entirely ruled out before the autopsy. The second great cause, and the most general cause in temperate climates, is appendicitis with abscess formation, even if the abscess be relatively small. In these cases there may be an ascending thrombosis and a preliminary pyelophlebitis or infection may occur through the retrocecal tissues—there is also the possibility through the lymphatic channels. The most common route, however, is by the portal vein, although the hepatic artery may carry the infection. Latent appendicitis, as in this case, is a cause, although much less frequent than when symptoms are present. Gastric and duodenal ulcers; typhoid and tuberculosis ulceration; infections of the gastro-intestional tract, including hemorrhoids and rectal carcinoma, are other causes. Gastric and duodenal abscesses may be a cause by direct perforation. Infection may originate in diseases of the pelvic organs, the spleen or the pancreas.

The causative organs are the usual pyogenic bacteria and especially B. coli and B. pyocyaneus. Abscess also may be contiguous from infection of the gallbladder or bile passages.

The case is much worth while from the viewpoint that a double abscess was present.

AN UNUSUAL CASE OF "VISCERAL CRISIS."

(Case 13302, Cabot Case Records, Boston M. & S. Jour., July 28, 1927.)

CASE HISTORY

A woman of thirty was brought to the Emergency Ward August 30. She stayed overnight in the ward. She had some fever. Next morning she was sent to the Out-Patient Department with a diagnosis of visceral crisis.

The only history was obtained in the Nerve Room. For four years she had had shooting pains in her legs at times. For a year she had had periodic attacks of vomiting lasting two weeks. During the year she had lost thirty pounds. Her feet and hands were numb. The day before admission she had difficulty in speech, was unable to say what she wanted to. There had been no sphincter disturbance. Examination showed tremor of the lips at times.

She gave a past history of scarlet fever and frequent sore throats. She had had marked scoliosis since a fall in childhood. She was sent that day to the wards. Clinical examination showed a much emaciated woman, semistuporous, but becoming quite violent when irritated. She was mentally confused and her speech was incoherent. The skin was dry and hot. There was a suggestion of right facial paresis. The spine showed marked left upper dorsal and left lumbar scoliosis with accompanying chest deformity. The left chest was flattened. The scapulae were angel-wing, more marked on the right. The lungs were clear and resonant. The apex impulse of the heart was felt in the fifth interspace 6 centimeters from the left of midsternum. The heart showed occasional extrasystoles and a loud rough middiastolic murmur over the apical area. Blood pressure 128/85. There was rather marked rigidity of the joints of the extremities. Right pupil smaller than left. Both dilated and slightly irregular. Right reacted sluggishly to light, left not at all. Accommodation not tested. Fundi normal. Biceps and triceps reflexes not well elicited owing to the patient's condition. Pronators normal and equal. Knee-jerks and ankle-jerks absent both in the ward and in the Emergency Ward. No true Babinski, although there was a suggestion in the left foot, and no true Romberg, although there was some swaying in the Emergency Ward.

Amount of urine not recorded, cloudy at all of three examinations, specific gravity 1.015 to 1.020. A catheter specimen showed diacetic acid, much acetone, sediment 2 to 5 leucocytes and 20 to 30 red blood cells per high power field. Culture from this specimen showed a slight growth of colon-like bacilli. Bliood normal except for 84 per cent polynuclears. Blood Wassermann negative.

A lumbar puncture in the Emergency Ward showed initial pressure 260, dynamics normal, fluid clear and colorless, ammonium sulphate strongly positive, alcohol positive, Wassermann negative, 72 cells, total protein 110, goldsol 0123211000.

Temperature 99° to 104°, rectal; pulse 87 to 160, respirations 20 to 40.

A medical consultant could find no pathology outside the spine. September 1 another lumbar puncture was done. The canal was entered between the fourth and fifth lumbar vertebrae. 25 cubic centimeters of clear colorless fluid was removed, initial pressure 275, dynamics normal, 15 cells, alcohol and ammonium sulphate positive, total protein 190, goldsol 1233320000, Wassermann strongly positive, sugar 41, chlorides 644. No tubercle bacilli found in sediment.

September 2 a firm rounded mass somewhat larger than a golf ball was felt in the abdomen just to the left of the umbilicus, freely movable upward, not connected with the bladder. The neck was slightly stiff. September 6 a third lumbar puncture gave 40 cubic centimeters of clear fluid, initial pressure 400, dynamics normal, 138 cells, alcohol and ammonium sulphate positive, total protein 138, goldsol 0123434110. Wassermann strongly positive; sugar not done, chloride 605, culture no growth. September 8 the patient died.

Discussion by Dr. A. C. Kingsley, Phoenix.

This "unusual case" of visceral crisis would seem to me to be rather typical until the day preceding her admission; at which time she had difficulty in speech and her illness entered its terminal stage. The shooting pains, periodical attacks of vomiting, numbness in hands and feet, pupillary disturbance, absence of reflexes, are characteristic symptoms of tabes dorsalis.

The history of scarlet fever and frequent sore throat is somewhat confusing. This, in connection with the mid-diastolic murmur at the apex, would suggest a possible heart lesion. However, she has never had any of the more characteristic symptoms of a serious lesion of the heart valves; consequently, I will take the medical consultant's statement that there is no pathology outside of the spine. Sore throat may have been the result of her luetic infection.

We now come to the day preceding her admission to the hospital, on which there was a marked change for the worse in her condition. She was semi-stuporous, confused, incoherent and violent if disturbed or irritated. This suggested definite cerebral involvement. The right facial paresis with the other localizing symptoms would attract us to the localized area at the base, where the seventh nerve might be independently involved.

The urinary examiation is not of particular interest, except the leukocytes and 20-30 blood cells with a positive colon-like growth on culture. It is possible that a colon infection of the kidneys might have accounted for some of the temperature.

Some explanation must be given for the mid-diastolic murmur; and admitting I am somewhat hazy on this point, I think perhaps it might result from a syphilitic aortitis.

Her first spinal fluid examination is fairly typical of tabes. The second and third indicate that there has been some change in the condition. The Wassermann is strongly positive. The protein is increased. The goldsol is more indicative of inflammatory process. The sugar and chlorides are reduced. The pressure was also increased. There was also some stiffness in the neck at the last examination.

I have no diagnosis for the small movable mass in the abdomen.

Differential Diagnosis:

Tuberculous meningitis. No bacilli were found; the fluid would be somewhat turbid; the sugar is low and the chlorides are markedly reduced.

Cerebral hemorrhage is possible, though the age of the patient and absence of evidence of vascular disease makes this improbable.

Cerebral embolism. If we knew more about the temperature, whether or not it was septic in character, we might give more thought to this condition. I doubt the presence of a septic endocarditis, which would be the natural source of an embolus.

Cerebral thrombosis with areas of softening. We have in this case, evidence of cerebral irritation as well as some focal signs. This condition is quite common in the early stages of syphilitic infection and mainly in persons under forty years of age.

Acute syphilitid meningitis. We have in this case positive evidence of syphilitic infection. The mental symptoms are those of an irritative lesion of the cerebrum; also some localized focal symptoms; some rigidity in the neck; the spinal fluid cell count is rather high for tabes. The colloidal gold is atypical, indicating a low grade inflammatory process.

There is a slight reduction in the sugar and chlorides. The protein is also moderately increased.

I believe therefore, the picture fits very closely from the clinical standpoint and an examination of spinal fluid—

Tabes dorsalis; the terminal stage of an acute syphilitic meningitis, with disseminated area of thrombosis in the cerebrum.

Discussion by Dr. T. T. Clohessy, Phoenix.

Our informant states that this woman had a visceral crisis, the "visceral crisis" in quotation marks. Visceral crisis means tabes dorsalis and tabes dorsalis means syphilis. The diagnosis of tabes dorsalis is stated by Osler to be justifiable if we find absence of ankle and knee jerks with one other sign, such as Argyll Robertson pupil. We have those three signs and many others to confirm them, such as four years of shooting pains in the legs at times; a year of periodic vomiting, each attack lasting two weeks; some numbness of hands and feet. At present pupils are unequal, dilated and irregular. This array of signs, significant in themselves, are reinforced by the spinal fluid examination, with all tests positive at first withdrawal except Wassermann. At the second lumbar puncture the Wassermann was strongly positive, which might have been the result of a provocative dose of arsphenamine. The number of cells was increased from 72 at first puncture to 136 at the third; this increase of cells, delayed in their appearance because of impermeability (relative) of the nervous tissues, is characteristic of the delayed meningeal Herxheimer or therapeutic shock, and tends to confirm me in my opinion that this woman has had some arsphenamine. The goldsol curve, high in the middle, also rises in height with each puncture, which is also characteristic of the Herxheimer reaction. So we must conclude that this woman without doubt has had a progressive tabes during the past four years at least. But patients do not die as a rule of tables per se, although death does at times occur from emaciation and exhaustion in gastric crises, with repeated and long continued vomiting. The diastolic murmur I would attribute to syphilitic aortitis. The urinalysis is not complete but points to an infective nephritis. The acetone we may attribute to the long continued vomiting with restriction of nourishment. The golf ball in the abdomen could be a pyloric or prepyloric syphilid, or a carcinoma. It is most likely a syphilid, as she is rather young for carcinoma, and, at any rate, there is not enough data to diagnose carcinoma. In fact, I consider the data rather incomplete for a definite diagnosis aside from the tabes dorsalis. However, I will venture, in addition, a diagnosis of pyloric or prepyloric syphiloma and acute infectious nephritis.

Discussion in part, by Dr. Clyde Marshall, (Massachusetts General Hospital, v. ibid.)

Everything in the physical examination would be consistent, probably, with neurosyphilis, not necessarily with tabes. She is stuporous and confused, which suggests that we may be dealing with taboparesis.

The goldsol is the first suggestion that all is not well with the diagnosis of neurosyphilis. The spinal fluid as given is not consistent with paresis. It is consistent with tabes or syphilis of meningovascular type. But to make a diagnosis of paresis we must have the whole five of the spinal fluid tests positive. We have, with an increased cell count, increased protein, a positive Wassermann, a paretic gold curve, and a globular ring. It is practically invariable, I think, that unless we have these five, we are unable to make a diagnosis of paresis. So that we shall have to look elsewhere for her condition of stupor and confusion.

Has she a meningovascular syphilis? The spinal fluid is consistent. The negative Wassermann does not trouble us much, but she is not likely to be comatose with only seventy-two cells.

In the second lumbar puncture the additional data of sugar and chlorides throws an entirely different light on the situation. It is still inconsistent with paresis, and also with syphilitic meningitis with coma, as we have only fifteen cells. The sugar is low, 41 milligrams per 100 cubic centimeters, and that can mean only one type of condition,—meningitis. It rules out other infections of the nervous system such as poliomyelitis, encephalitis, brain abscess or sinus thrombosis. All these would have a normal or high sugar. The chlorides are also low. Normal spinal fluid chloride is 720 to 750 milligrams NaCl per 100 cubic centimeters. Low chloride occurs chiefly in meningitis. It can occur in poliomyelitis, which however does not come up here, and could be otherwise ruled out by the sugar. So these two findings point toward the diagnosis of meningitis of some sort, of which no previous indications are given in the history. It is true she has had some "rigidity of the joints of the extremities." It does not mention particularly a Kernig or head retraction. But nevertheless a spinal fluid such as stated could mean meningitis of some sort and nothing else. It however leaves doubt as to whether she could have had a previous syphilitic condition.

The findings in the last lumbar puncture were essentially the same as those in the other except that the chlorides have gone lower this time, to 605. They go as low as that with practically nothing except tuberculous meningitis. A chloride of 605 is practically pathognomonic of that condition.

The question of syphilis has to be considered on the clinical history. She had four years of shooting pains in the legs, also of vomiting, which is suggestive of visceral crisis, which diagnosis was made in the wards, I think it is highly probable, although we have no more data than that, to assume that she did have central nervous system lues. The Wassermann in the spinal fluid would suggest it, but in the presence of meningitis, and with a positive blood Wassermann, one is not absolutelly safe in saying neurosyphilis; because according to Greenfield, with a meningitis and a positive blood Wassermann, the blood Wassermann may spill over into the spinal fluid and give a positive Wassermann without definite evidence of neurosyphilis. In this case we have only the blood Wassermann report, which is negative. Hence with the meager history we can only say we think she may have it.

I think we can make a diagnosis of tuberculous meningitis, most probably also tabes. The question of the heart is hardly within my field. She has a rough middiastolic murmur, with a history of fever and sore throat. Are we entitled to make a diagnosis of mitral stenosis on that?

The question of the golf ball inside the abdomen is entirely beyond me also. From a neurological point of view we would say tuberculous meningitis, probably neurosyphilis, and if so probably of tabetic type.

Discussion by Dr. Tracy B. Mallory.

One point that is not brought out very well in the physical examination, it seemed to me, was the woman's extreme emaciation. The circumference of the thigh at its greatest point was only twenty-four centimeters. The arms were so thin that a child

could have encircled them with its fingers. There was also an extreme scoliosis.

The lungs showed a few quite small caseous areas, and also a few older calcified ones. The bronchial glands showed an occasional calcified spot. The heart weighed 200 grams. The measurements of the valves were normal, but the mitral valve showed very definite thickening along the margins, and also some thickening of the chordae tendineae, I think sufficient to give the murmurs recorded in the history, but probably causing no symptoms, and its small size was due to the general extreme emaciation of the patient.

The liver was also very small, weighing only 850 grams. Section of the inferior surface showed numerous miliary pinkish white foci. The kidneys showed one fair sized caseous area four millimeters in diameter. The most important tuberculous focus, other than the acute terminal miliary condition, was a fairly large tuberculous ulcer of the cecum, evidently of quite long duration, as the lymph nodes immediatelly draining that area showed evidence of involvement of months' to perhaps years' duration.

The meninges showed a very fine sowing with minute tubercles, particularly in the arachnoid over the superior surfaces of the temporal lobes. It was still extremely early meningitis, but there is no question of the diagnosis. The main part of the brain has not been sectioned, so that we have no proof whether there was neurosyphilis or not.

Q. What was the mass in the lower abdomen?

Ans. There was none.

Q. Were there any foci in the vertebrae"

Ans. That was what we naturally guessed, but cross sections of the lumbar vertebrae did not show any old areas there. I think that the scoliosis must have been postural and not tuberculous.

Q. Doesn't tuberculous meningitis give a positive Wassermann?

Ans. I think that the Wassermann is extremely unreliable in any case of meningitis. I have seen it positive a great many times when I did not think there was any evidence of syphilis. Without the best history of syphilis I should certainly discount a positive Wassermann practically entirely in the presence of meningitis.

ANATOMIC DIAGNOSIS

1. Primary fatal lesions.
   Tuberculous meningitis.
   Tuberculosis of the lungs.
   Miliary tubercles of liver and kidney.
2. Secondary or terminal lesions.
   Central necrosis of liver.
   Tuberculous ulcers of cecum.
3. Historical landmarks.
   Scoliosis.

PERSISTENT COUGH OF UNUSUAL ORIGIN

(Case 16182, Cabot Case Records, N. E. Jour. of Med., May 1, 1930.)

CASE HISTORY

An Irish watchman sixty-five years old entered the hospital complainnig of persistent cough of about six weeks' duration.

He had never known what it was to be sick before the present illness. He had never had any symptoms pointing to chronic pulmonary infection or any dyspnea on exertion. He had had no gastro-intestinal symptoms at all. He was constipated as a rule, but there had been no change in his bowel habits recently. About six weeks before entry he had sudden onset of a cough which lasted a few minutes at a time. At times he raised a small amount of sticky yellow sputum,—practically an unproductive cough. After a week of this without chills, fever or night sweats he gave up work and rested at home for a week without improvement. He then went back to work for another week, but felt so tired and unrelieved of his cough that he gave up work entirely three weeks before entry. Since that time he had not improved at all but had increasing orthopnea and anorexia and lost ten or fifteen pounds in weight. Three or four nights previous to entry he slept very little, craving the air. He spent much of each night sitting by a window. He had had no pain on the left side of the chest, but a week or two preceding entry the cough became definitely painful, the pain being referred to the lower right chest.

When seen here he proved to be all skin and bones, a little old man sitting upright in bed panting for air. On examination he proved to be cyanotic. His heart was not remarkable in size. There were no significant murmurs. The rate was about 100. The chest signs were interesting. Over the right lower lobe in the back there was diminished tactile fremitus with diminished breath sounds. There was no bronchial breathing, no egophony, no difference between the right and the left chest on inspiration. No peripheral edema, no enlarged liver. Blood pressure 130/70. Some secondary anemia. Leukocyte count 15,000 to 16,000.

He was given one-sixth grain morphia. We tried to keep him as comportable and as well as possible. At midnight the day of admission he was found to be in even worse condition, with more air hunger than he had before and his chest full of rales. Venesection was done, which seemed to relieve his dyspnea and cyanosis. His pulse however did not become any less at that time; it was 120 to 130, at no time going below 120. By morning his chest had cleared so that the signs were much the same as on entry. On morphine given at least once every three hours, digitalis and rather frequent caffeine he had a stormy day, with his paroxysmal cough continuing. He had a great deal of difficulty in getting his breath. The pulse continued 140 to 150. The blood pressure stayed up pretty well, the systolic not below 115 throughout the day. He continued to be quite cyanotic. No irregularity in the heart. An electrocardiogram showed no changes that would not be compatible with his age. We had a portable chest plate that day. During the night he continued in about the same state. At four in the morning the blood pressure fell to about 80 or 90 systolic. The heart rate continued at 140 to 150. He petered out during the next four hours. Venesection was of no avail. We thought that the physical signs were due to a diminished aeration of the right lower lobe, and that there was no fluid.

The x-ray films show dullness at the right base which has the appearance of fluid obliterating the outline of the diaphragm. It rises somewhat higher in the median than in the axillary line. The costophrenic sinus is obliterated. The heart is not particularly enlarged and apparently not displaced. In addition we have this coarse, rather soft mottling scattered throughout the lung fields. There is dullness at the right apex. That might be part of the same process that is present at the base, that is, fluid. The plates were taken with the patient lying down. The process in the lung is of a bronchopneumonic character and could be due to a number of things. We see appearances like this in malignancy, also in heart failure. I do not attempt to say what the process is due to here. (Holmes).

Discussion by Dr. S. I. Bloomhardt, Phoenix

The important points in this history are cough, orthopnea, weakness, and cyanosis in a man of 65. He had been apparently perfectly well until these symptoms began, and he rapidly became worse in spite of treatment and died six weeks later.

In discussing this case, we would first like to mention some of the common causes of dyspnea and cyanosis. Pulmonary tuberculosis is, probably, the most common cause, but as this man was perfectly well up to the time of the beginning of these symptoms and this condition only lasted six weeks, we feel like we could rule out pulmonary tuberculosis. Cardiac disease; it is mentioned that there was no peripheral edema and we do not believe that one could have such a marked orthopnea and cyanosis from heart disease without some edema of the extremities; also, the x-ray picture and the electrocardiogram did not reveal any abnormalities of the heart. Sometimes, there are marked changes in the heart that are not revealed by x-rays or other examinations. It is said that this man was extremely emaciated. With such emaciation, our first thought would be tuberculosis or a malignant disease. Could there be a milignancy of the lung, Six weeks seems to be entirely too short a time for malignancy to cause such severe symptoms. A pneumothorax causes cyanosis and dyspnea but there is no history of a sudden onset or shock that is usually experienced in a spontaneous pneumothorax. A foreign body in one of the large bronchi could cause these symptoms, but there is no history of such a condition. A new growth pressing on the trachea or main bronchi might also cause these symptoms but we do not believe there is sufficient evidence to warrant this diagnosis. Miliary tuberculosis may be so acute as to cause death in one or two weeks. It is usually observed in three types,—the cerebral and typhoid types are the most common and the pulmonary type less frequent. In the pulmonary type of miliary tuberculosis, we have an extreme dyspnea entirely out of proportion to the amount of lung apparently involved. There is also, usually quite a marked cyanosis; also, an irregular temperature. The leukocytosis, the day before death was probably due to a terminal infection. There was, no doubt, quite a marked weakness of the heart as the rate came faster each day and the systolic blood pressure became lower. This could be accounted for by a weakened heart muscle due to toxemia and insufficient food supply. The history in this case is not complete enough to make an exact diagnosis. We believe it is either a disseminated malignant disease of the lungs or an acute miliary utberculosis of the lungs of the pulmonary type. If there was no fever throughout the disease we certainly would not suspect miliary tuberculosis; also the age would be against tuberculosis. My colleagues lean to the diagnosis of malignancy. I will place my vote on miliary tuberculosis. If, however, there was no fever, I would then think it is a disseminated malignancy of the lungs.

Discussion by Dr. Victor Randolph, Phoenix

Chronic cough in a man 65 years old is most often due to chronic bronchitis which in a good many cases is secondary to cardiac conditions. In this case it makes us think of latent bronchitis or bronchiectasis, chronic cardiac weakness which has reached the stage of failure or of acute bronchitis or of some chronic latent infection such as tuberculosis or mycotic disease, malignancy or syphilis.

The physical examination apparently rules out any cardiac condition except hypertension which has recently reached a lower level but the electrocardiograph rules this out also. We have then some pulmonary condition as the cause. We can now say that this record as a whole illustrates very well the type of study which seems to have been made in a great many of the pulmonary cases which are referred to Arizona by eastern physicians. There is no record of sputum examination. There is also in this record no temperature findings. It is conceivable that this man had acute bronchitis which developed during a period of six weeks into bronchopneumonia. It is also possible that he had latent pulmonary uberculosis which developed into a bronchopneumonic type. However, in the absence of any temperature record and sputum examination, we can say nothing further. We are forced to the conclusion that he has an obstructive lesion in the right lower bronchus which is sufficient to give physical signs and x-ray signs. The fact that the x-ray densities extend higher along the medial portion of the right chest than laterally is in favor of malignancy more than of fluid that may be present. The other densities in the chest which were interpreted as bronchopneumonic may well have been metastatic malignancy. We can consider this whole picture as primary malignancy in the right lower bronchus although we cannot entirely rule out the possibility of a primary source in the rectum or some other small primary source. This of course is unlikely because of the absence of abdominal masses. We may find malignancy with terminal pulmonic patches. As a second diagnosis we would consider simply broncho pneumonia with long standing hypertension.

DIAGNOSIS

(1) Carcinoma of right lower bronchus, with metastic malignancy of lungs.
(2) Bronchopneumonia.
(3) Tuberculosis.

Autopsy Discussion by Dr. Tracy B. Mallory
(Massachusetts General Hospital, v. ibid.)

This case is in many ways one of the undiagnosable. The primary disease was cancer of the stomach. I think there is no question that his major symptoms were due to pleurisy on the right side. At autopsy 1400 cubic centimeters of frankly bloody fluid was found there. The story of six weeks' unproductive cough and dyspnea means that the fluid was there all the time. We were much interested at the time of autopsy because we could not find any gross evidence of tumor in the pleura or in the lung. But when the sections came through there were microscopic emboli of tumor in vessels and lymphatics throughout the lung. I am pretty sure that if one had looked at the blood smear very carefully some tumor cells would have been picked up in that.

Both lungs are involved though the process is a little more marked on the left. The physical signs in this case are of interest and unquestionably misleading. I have a very vivid recollection of a case I saw as an interne which had every possible sign of consolidation, including increased tactile fremitus. The story, however, was that of pleurisy, and when we put in a needle 1800 cubic centimeters of fluid was withdrawn.

The tumor in the stomach was a partially annular tumor encircling about two-thirds of the stomach, causing partial but not very great obstruction.

ANATOMIC DIAGNOSIS

1. Primary disease.
   Carcinoma of the stomach.
2. Secondary or terminal lesions.
   Multiple microscopic tumor emboli of the lungs.
   Carcinomatous pleuritis.
   Hemothorax, left.

JAUNDICE AND ASCITES IN A BOY OF EIGHTEEN

(Case 14281, Cabot's Case Records, New Eng. Jour. of Med., Aug. 20, 1928, page 435).

Case History

An American schoolboy of eighteen entered the hospital September 28 with a diagnosis by his physician of cirrhosis of the liver.

A year and a half before admission when in apparently good health he woke up at night with a very sharp pain in the abdomen localized below the umbilicus, more on the right. It doubled him for twelve minutes, then gradually wore off. He stayed in bed for two days. There were no clay colored stools or jaundice. From thirteen to seven months before admission he had gas (heartburn) relieved by white powders given by his physician. A year before admission after a cold in the head a physician found a slight infection of the gallbladder with some jaundice. He had dull generalized headache when walking, but no other pain. He was in bed a week and recovered completely in three weeks. His urine was dark. During the next six months he was not quite in his usual health and had three similar attacks, two of them just before the holidays, when he had been eating too much candy. As the last attack was subsiding, six months before admission, he went to a physician, who found a severe bronchitis which lasted three weeks. There was also an acute ear. The liver was somewhat enlarged. There was more or less continuous right sided pain along the eighth and ninth ribs relieved by lying on his right side, possibly pleurisy. He vomited once. The vomitus contained dark material resembling old blood. At the onset of the next attack, three months before admission, his physician found an enlarged liver, ascites and edema of the feet and legs. The temperature was 101° to 105° for three weeks. During this period there were night sweats. All the symptoms then disappeared. Two weeks before admission the patient had a severe cold followed by symptoms similar to those of the last attack. These symptoms persisted. With the cold he had large glands in the neck which had subsided at admission. He had been on a skim milk diet with sodium phosphate enough to keep his movements watery. He had vomited only once; blood in the vomitus was questionable.

One grandfather died of cancer. The family history is otherwise not important.

The patient had had no serious diseases, but had always been in perfect health and very strong until the present illness. Six months before admission he had a questionable pleurisy. Four months before admission he had a purulent discharge from the right ear which cleared up. He urinated once or twice at night. At admission his mouth was sore, possibly from a wisdom tooth. Six months ago he weighed 140 pounds; his present weight was 142.

Clinical examination showed a weak, wasted, ill-nourished boy with dry and jaundiced skin, jaundiced sclerae and sparse hair, unable to sit up. The tonsils were enlarged, cryptic, and filled wtih cellular debris. There were pea-sized axillary glands. The chest was flat. The ribs were prominent. The apex impulse of the heart was not seen or felt. There was no enlargement to percussion. The sounds and action were normal. The pulmonic second sound was accentuated. There was a systolic murmur in the left third interspace. Blood pressure 100/50 and 30. There was some dullness at the bases of the lungs behind. The abdomen was tense and shining, with shifting dullness, very marked fluid wave and an air bubble. The liver edge was felt by ballottement three finger-breadths down at least, slightly tender. The spleen was felt in the same way three fingers down. There was slight umbilical hernia. The scrotum was filled with fluid and enlarged to the size of an orange. Both legs showed pitting edema to the thighs. The pupils were normal. The knee-jerks were obtained on reinforcement.

Before operation urine normal in amount, specific gravity 1.040 to 1.020, cloudy at 4 of 6 examinations, alkaline at all, the slightest possible trace to a very slight trace of albumin at 5, bile in 6 of 7 specimens, 0 to 1 leukocyte and 0 to 1 red cells per field in one of six specimens of sediment. Blood: 3,450 leukocytes, 52 per cent, polymorphonuclears, 24 per cent, lymphocytes, 12 per cent, large mononuclears, 12 per cent unclassified. (Differential on a basis of 25 cells.) Hemoglobin 45 per cent., 3,000,000 to 2,600,000 to 3,100,000 reds, achromia, slight variation in size and shape, no stippling or polychromatophilia. Platelets decreased. Reticulated cells 1 per cent. Icteric index 50 at admission, 20 Ocotber 6 and 12, 18 October 17. Bleeding time 8½ minutes to 4½ minutes. Clotting time 7 to 9 mintues. Retraction normal. Fragility test began at 44, complete at 28. Wassermann negative. Van den Bergh test direct, positive, quantitative 8.8 miligrams per 100 cubic centimeters. Rosenthal bromsulphalein test: after 30 minutes 20 per cent. Cholesterol (serum), .084 per cent. Stools: guaiac negative at 3 examinations, bile indefinite at 2.

Until October 12 temperature 97° to 101°, then 98° to 103° until operation. Before operation pulse 78 to 121, respirations 12 to 32.

The day of admission an abdominal tap gave 4,000 cubic centimeters of clear bile stained fluid, specific gravity, 1,008, 500 red cells and 100 leukocytes, mostly lymphocytes; culture, no growth. Immediately after the tap the liver edge was felt two-thirds of the way down to the umbilicus, extending well to the left. The edge was sharp and slightly tender, and appeared grossly irregular, as if the liver were lobulated. The spleen was felt 2 centimeters below the costal margin. The patient was more comfortable after the tap.

An x-ray plate taken October 4 after a barium meal showed a very large gastric outline. The stomach was apparently distended by gas. The outline of the transverse colon was also visible and appeared to be filled with gas. The diaphragm was high on both sides. Just above it on the right there was a triangular area of increased density with very sharply defined margins, possibly an area of atelectasis of the lung. Otherwise the examination of the chest was negative. There was nothing unusual in the appearance of the heart shadow.

The jaundice cleared rapidly and by October 7 was nearly gone. Distention was now the chief complaint. The fluid in the abdomen reaccumulated. The patient was symptomatically better.

October 21 a fluoroscopic examination of the stomach without barium showed a large amount of gas in this region. The colon was also distended with gas as in the previous examination.

After a number of consultations operation was done October 22. After the second day the temperature came down to normal. There was moderate distention relieved by enemas. The wound was in good condition. The patient however did not look very well. The temperature was 100° October 29 and after this ranged between 99.2° and 101.9°. October 31 the patient was much worse,

irrational part of the time, with very poor nutrition. Intravenous glucose was given without result.

November 1 blood examination showed 9900 leukocytes, hemoglobin 60 per cent., 3,300,000 red cells fairly well filled with hemoglobin, considerable variation in size, but only slight poikilocytosis; platelets normal. A count of about 500 cells showed no lymphocytes, 99 per cent. polymorphonuclears, a few immature and a few old but no more than a normal smear would show. No smudges were made out.

Discussion by Dr. Frank J. Milloy, Phoenix.
(Phoenix Clinical Club, Apr. 14, 1930).

The best way to approach the diagnosis in this case is to consider the main symptom and physical finding, and that is ascites. Eighty-eight per cent of the cases of ascites are caused by cardiac or renal disease, hepatic cirrhosis, or peritoneal tuberculosis, in the order of frequency named. The other twelve per cent are caused by malignancies, anemias, adhesive pericarditis and various other unusual conditions. This history and physical findings do not suggest very strongly a cirrhosis of the liver, either biliary or atrophic. Neither is it due to renal disease. It could very readily be due to cardiac disease, but cardiac disease is not operated upon, so that it cannot be cardiac disease alone, although there is no doubt some myocardial weakness present and some cardiac decompensation which is probably secondary to the primary disease. This though there is no doubt some myocardial weakness present and some cardiac decompensation which is probably secondary to the primary disease. This leaves, as the most likely cause, peritoneal tuberculosis, or some of the less frequent causes of ascites.

The history of onset in this case , with a more or less continual train of digestve symptoms, the irregular periods of temperature, the age of the patient, all strongly suggest peritoneal tuberculosis. The loss in weight, the drop in blood pressure some glandular enlargement occur in tuberculosis; also the discharging ear. The character of the ascites is not typical of tuberculous effusion in the peritoneum but it does occur with as low a specific gravity as this. The prevalence of lymphocytes and the absence of growth on culture fits with a tuberculous effusion. The x-ray examinations, showing the stomach and intestines more or less distended with gas is what one would expect with peritoneal involvement. The secondary anemia and marked leucopenia, and high lymphocyte count fit in exactly with peritoneal tuberculosis. But there are still many symptoms unexplained. The blood picture is identical with that which occurs in Banti's disease, and that condition would explain the jaundice also. But the spleen is hardly large enough, since Banti's disease produces one of the largest spleens that occur in medical practice. The absence of blood in the stools and absence of hematemesis is against Banti's disease as well as cirrhosis of the liver. If the patient had had Banti's disease, the operation could have been for the removal of the spleen. There is a marked similarity between Banti's disease and Hodgkin's disease and this patient could have had an abdominal Hodgkin's but if this were true, a serious error occurred when the patient was operated upon.

Adhesive pericarditis will explain more of the physical findings in this patient than any of the above conditions. You could expect an anemia but it would be very unlikely to have a leucopenia and a lymphocytosis. The generalized edema and ascites and enlargement of the liver and spleen could all be due to failing myocardium, which could be secondary to tuberculous peritonitis, Banti's disease, or any other long standing sickness. But with the other cardiac findings, it is very probable that the boy had an endocardial involvement. The accentuated second pulmonic and systolic murmur strongly suggest mitral disease. The high pulse pressures of 50 and 70 and the low diastolic pressure of 30 are almost certain to be aortic disease. The preliminary history of this patient could have been caused by a low grade infective endocarditis. Such an infection would explain the enlarged glands in the neck which subsided later. If they had been tuberculous glands they probably would not have subsided. The infective endocarditis could have caused this valvular disease and then could have produced a pericarditis resulting in an adhesive form. The inability to see or palpate the apex impulse of a heart as decompensated as this heart would be compatible with an adhesive pericarditis, and it would be hard to explain it in any other way. The jaundice was probably caused by pressure in some way on the bile ducts, as it subsided as soon as the pressure was released. The operation was performed to relieve the pericardial adhesions. In the absence of a blood culture and the report of a guineapig inoculation with the ascitic fluid, both of which have probably been purposely omitted to make the case interesting, it seems as if some guessing will have to be done in deciding on a diagnosis. I think it rests between tuberculous peritonitis, Banti's disease, and adhesive pericarditis with an associated endocarditis; a possible fourth condition is abdominal Hodgkin's. All the first three are mostly likely to occur at this age and all three are amenable to surgical treatment. In the absence of a very large spleen, the blood picture is the only thing that is typical of Banti's. Absence of hematemesis and negative guaiac tests are against it. Negative lungs, negative history and negative other foci of infection, are against tuberculosis, although the boy could contract the bovine type through the intestines. But peritoneal tuberculosis, while it will explain the blood picture and ascitic fluid, hardly explains all the cardiac symptoms and findings. If we can disregard some of the laboratory findings and make a diagnosis on the symptoms and physical findings, I think it will be,

Adhesive Pericarditis
Chronic Endocarditis (rheumatic type).

Discussion by Dr. O. H. Brown, Phoenix.

The high points in this record are: A boy, 18 years of age entering the hospital, gave following history. One and one-half years ago he had an acute attack of pain in right middle abdomen, apparently appendicitis. For about six months after the appendicitis he had heartburn and gas. A year before he had a gallbladder infection with jaundice and dark urine, followed in next few months by three similar attacks. Shortly before admission he had bronchitis, ear discharge and pain along the right lower base of the lung. He vomited a material which looked like blood. Three months before admission he had enlarged liver, ascites and edema of the feet and legs with temperature up to 105. He had been subject to nocturia. He was a weak, wasted, ill-looking boy with a dry jaundiced skin and sclerae. Tonsils were lalrge and diseased. Axillary glands were pea sized. Systolic murmur in the left, third interspace. Diastolic blood pressure as low as 30, systolic 100. Spleen was enlarged. The icteric index was as high as 50 per cent. There

was leukopenia and moderate anemia. The direct Van den Bergh was positive. The Rosenthal was 20 per cent. Fragility test was normal or a bit better than normal. The ascitic fluid was withdrawn and the liver was found to be markedly irregular as though lobulated. Stomach was much enlarged. Colon was distended. A small indurated area in the base of the right lung was found; edges were sharply defined. The diaphragm was high. Patient was operated upon. From this history it seems definite that the patient had a large liver and a large spleen with obstructive jaundice. There was also evidence of obstruction to the outlet of the stomach. Probably also something was interfering with the passage of fecal material through the intestines as the colon was much dilated.

The enlargement of the liver and the spleen together with the anemia makes one think of some type of splenic anemia. Cirrhosis of the liver, however, may explain the picture even though cirrhosis more commonly occurs later in life. Enlargement of the spleen suggest Banti's disease but splenic enlargement also occurs in cirrhosis. In cirrhosis, however, we ordinarily get leukocytosis. In Banti's disease there is an increase of fragility of the corpuscles. In this case the fragility test was normal. The edema and the jaundice may both be explained by a severe grade of portal cirrhosis. Likewise the heart murmur may be explained by the advanced grade of cirrhosis. It is very common for a contracted liver to be nodular in cirrhosis but this liver was large.

Hodgkin's disease may affect any part of the body though the common place is in the neck, on one or both sides. It may produce extensive edema and interfere with circulation and may obstruct other organs. It is also common to see a large spleen with Hodgkin's and it does produce an enlargement of the liver. Leukopenia is the rule. The fragility test is not greatly altered.

My guess at the diagnosis then is Hodgkin's disease of the abdomen producing obstructive jaundice and edema and nodules in the liver and enlargement of the spleen; moderate cardiac dilation; secondary anemia.

Discussion, in part, by Dr. Richard C. Cabot.

Cirrhosis of the liver is a very surprising diagnosis at that age..

Tuberculous peritonitis is possible. There is also a case I happen to remember here, a chronic pericarditis working through the diaphragm and to the capsule of the liver, with actual cirrhosis.

Some of that increased weight was presumably due to ascites and edema.

The main thing we have to bear in mind as we come to the physical examination is that the boy had ascites, apparently enlarged liver and some previous attacks with fever and symptoms that sound like gall bladder inflammation. Of course gall-bladder inflammation cannot account for enlarged liver and ascites. He may perfectly well have had both, but he could not have the latter as the result of the former. No one has said anything about the spleen, or of the possibility of the only type of neoplasm seen at this age often, that is the leukemic. He had some enlargement of glands in the neck, but it subsided. We must remember the possibility of tuberculous peritonitis. But I am quite at sea prior to the physical examination.

That systolic murmur does not mean anything in a boy with this clinical picture.

With a big liver dullness at the bases of the lungs is probably due to a high diaphragm, not to anything wrong in the chest.

"The abdomen was tense and shining, with shifting dullness, very marked fluid wave and an air bubble," that is, the intestines floating up in the epigastrium.

I do not see anything about sugar in the urine report, but I assume that it is abesnt. Of course in an emaciated boy we naturally would wonder if it had shown in the urine.

The blood shows a secondary anemia. The chief think about the blood is the extraordinarily low leukocyte count.

Differential diagnosis: It seems to me we have to say he has a cirrhosis of the liver, as they said in the beginning. I do not know what else would give such a chronic portal obstruction as we have evidence of here. The tap fluid is enough to throw out tuberculous peritonitis. After such a tap if there were any malignant disease we ought to be able to feel it.

We are not given any evidence of a chronic pericarditis. When you put these things aside I do not know anything that is left to cause such an ascites as this except a chronic interstitial process in the liver. What type can it be? I do not see any probability at all that it is the usual type which most of us associate with alcohol. Syphilis of course comes to our minds when we hear that the liver edge was lobulated. But we have nothing else to back up that diagnosis. Syphilis of the liver at this age is unusual. We do not usually have so much ascites. We often do not have jaundice, and I never knew it to kill anybody without other troubles.

Acute yellow atrophy or subacute yellow atrophy, toxic hepatitis, I can not exclude. We seem to have a big liver, and that, if it is correct, would exclude it. There may be stages of a toxic hepatitis without any diminution in the size of the liver. But we should not expect it to go on to death without diminution in its size. That is the chief evidence against it; but I do not feel able to exclude it. He has had two infections, one in the abdomen and one in the ear. It might be an infectious type of cirrhosis starting from a cholangitis and spreading up from the bile passages.

His anemia is an outstanding thing. We have to consider this as a hemolytic type of anemia and the hemolytic type of jaundice, connected with some abnormal activities in the spleen. I have not much doubt that the operation done was splendectomy for the removal of trouble supposed to be of that type. Between that and the infectious type of cirrhosis I do not know any way to make a clear differentiation.

Banti's disease is one of the most cloudy clinical entities that we have. I think the diagnosis cannot be made unless we prove first that we have an enlarged spleen and later a cirrhotic liver. It cannot be excluded here, but I cannot say anything in its favor.

I see no reason to consider Hodgkin's disease. We hear of cases that are confined to the spleen and liver, but we should have no evidence outside that, especially when we have such a very low white count. I should think we had better not. I have no idea how to explain that differential count of 99 per cent. of polymorphonuclears. I have never seen so high a count. I feel rather skeptical.

PRE-OPERATIVE DIAGNOSIS

Infectious jaundice.

OPERATION

The abdomen was found to be filled with clear fluid. The liver was very much enlarged, perfect-

ly smooth and instead of being dark was rather speckled in appearance and lighter in color than normal. A small piece was taken out for microscopic study. The spleen was much enlarged, about eight inches in length, much thickened and also felt smooth. The gallbladder was much thickened and edematous. In the retroperitoneal tissue above the pancreas there seemed to be more or less thickening or edema, as the tissue was thick enough to prevent the feeling of the normal structures. A tube was put into the gallbladder.

DR. MALLORY: The microscopic findings were a complete surprise to everybody. The liver showed a very marked infiltration around all the portal areas of large, immature, abnormal lymphocytes showing very numerous mitoses. The histologic appearances were typical of lymphatic leukemia. I think the only possible diagnosis is an aleukemic lymphatic leukemia.

In leukemia of this type the lymphocytes are very abnormal. I suppose the explanation is that they are not sufficiently differentiated to live in the circulating blood stream. They are essentially tumor cells; no normal lymphocytes are being produced at all.

DR. CABOT: Should you assume that we should have found something in the marrow?

DR. MALLORY: Yes. I believe all the organs would be similarly infiltrated.

HISTOLOGIC DIAGNOSIS

Lymphoblastoma.

ENURESIS

Review of Current Literature

ORVILLE HARRY BROWN, M.D. Ph. D.
Phoenix, Arizona.

(Read before the Maricopa County Medical Society, October 29, 1930).

Enuresis according to the dictionary is involuntary emptying of the bladder.

Adrien Bleyer[1] defines enuresis as a disturbance of the physiology of micturition in which the physiologic control of the brain is blocked by stronger stimuli which have to do with the nervous mechanism of the bladder. Enuresis vear is a term he suggests to indicate the type where there is disassociation of the voluntary nervous system from the act of micturition; and pseudo-enuresis he uses for the type in which influencing the will relieves the condition.

According to Israel S. Wechsler[2] incontinence may develop in tabes, combined sclerosis, myelitis, tumors of spinal cord, cauda equina lesions, hematomyelia of lumbar cord, injuries to cord, and spina bifida.

M. B. Wesson[3] and H. H. Young[4] made anatomic and embryologic studies which indicate micturition is ordinarily an act of the will upon the trigone of the bladder against the weak sphincters which are under the control of the involuntary nervous system.

D. I. Macht[5] took advantage of the selective actions of epinephrin and ergotoxin on the sympathetic system and of pilocarpin and atropin on the parasympathetic and found that the innervation of the trigone of the bladder is independent of the rest of the bladder.

Chetwood[6] savs in children the detrusor muscles of the bladder are relatively thick and strong as compared with the spincters, and therefore the bladder may empty with great ease.

H. H. Young[7] thinks relaxation of the bladder sphincter is not an act of inhibition but is brought about by the muscles of the trigone.

John McK. Mitchell[8] makes the point that throughout childhood the diet is primarily liquid and hence there is a relatively large quantity of urine calling for frequent emptyings of the bladder. He says it is often as late as the fourth to fifth year before the parents become duly impressed that the bed wetting is an abnormal habit. He also says polyuria is a cause in certain cases, sleeping cold may cause excessive diuresis, and excessive intake of fluid, diabetes mallitus and insepidus and nephritis, etc. may be factors.

Grover[9] studied 200 cases of enuresis and found in the family a history of it in 56 per cent of the cases.

Theodore H. Weisenburg[10] reported upon an investigation into enuresis by Forrest N. Anderson who spent a year on the problem, "diurnal bladder control is secured at an average age of 17 months, nocturnal bladder control at an average age of 23 months, with an average training period of six months." He says "the infant bladder is ordinarily of sufficient size to hold the night's secretion of urine; fluid restrictions are of no great value; emotional factors are the predominant factors in causing or at least in continuing enuresis." Physical factors may be of importance especially from a suggestive angle.

Ralph C. Hamill[11], basing his ideas on an extensive experience, concludes that enuresis is a conduct disorder; that it is of prime importance that the child should assume responsibility for its conduct in its sleep; and that all other forms of treatment are against the child's interest. In a group of 55 cases who properly followed treatment 72 per cent recovered. It is Hamill's view that all persons to greater or less extent make themselves responsible for certain acts during sleep; nearly all react during sleep to stimuli of micturition; many persons can waken at definitely specified hours, etc.

K. M. Horton[12] had a series of 60 cases and the majority occurred after control of micturition had once been established. Practically 50 per cent had no physical abnormalities; one case only had phimosis. It was most common for enuresis to be both diurnal and nocturnal. He says, "the children were in most cases nervously unstable, highly strung persons, abnormally sensitive to praise or blame" and that many cases exhibited home incompatabilities. All cases responded to suggestive measures in combination with giving the belladonna.

Basil A. Hayes[13] discusses the bladder and urethra abnormalities which cause incontinence. He writes: "The sphincters of the bladder consist of two muscles, one a weak voluntary circular and the other involuntary longitudinal fibers which pull on the anterior portion of the bladder neck. They are assisted by the levator and muscles which lift up the entire bladder floor.

The expulsive force of the bladder is not sufficient under ordinary circumstances to force open these sphincters. There is provided also an opening muscle which lies on the trigone and runs through the orifice of the bladder neck. Voluntary contraction of this muscle pulls open the closed sphincter.

True incontinence can only occur if this muscle remains in a constant spasm or if the sphincter loses its tone and remains open. Even if the first of these occurs, the external sphincter will hold the stream.

Ulceration, tumors, nerve lesions, sagging of bladder floor, trauma, and congenital defects are all

(Continued on page 602)

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PHOENIX NEWSPAPER COMBINATION

The purchase of the Gazette by the Arizona Republican, at Phoenix, Ariz., was a nine days' topic of comment, and the cause of many congratulations to the owners of the Republican,—recently changed to the Arizona Republic. Mr. Charles Stauffer invited about three hundred representative citizens to a dinner at the Westward Ho, and made announcement of this purchase and the combination of the two papers under one management, amid scenes of great enthusiasm.

The phase of this combination which will be of special interest to the medical profession will be the policy of the papers with regard to advertisements. Many years ago the Republican adopted the policy of the most advanced newspapers, of refusing its advertising columns to all quack remedies and all medical or surgical appliances of questionable merit. This has not been the policy of the Gazette in times past, but it is anticipated that this will be changed in the future. Some of the advertisements still being carried will not measure up to the standards of the Republic, but doubtless these are being carried on contracts still in force and it is expected that both papers will be placed on the same high plane, so far as advertisements are concerned, as they have been in their news and editorial columns.

We welcome this combination and congratulate the owners of the Arizona Republic on their courage and farsighted wisdom.

FINISHING OUR FOURTEENTH YEAR

This issue closes the fourteenth year of publication of SOUTHWESTERN MEDICINE. Since there has come to our attention recently evidence of misconception, in otherwise well informed circles, of the relation to this journal of the various organizations interested in it, it seems appropriate to set forth, again, the essentials of the business organization.

SOUTHWESTERN MEDICINE is owned jointly by four organizations, namely: the El Paso County Medical Society, the New Mexico State Medical Society, the Arizona State Medical Association, and the Medical & Surgical Association of the Southwest. Each of these organizations selects from its membership two members of a Board of Managers. This Board elects its own chairman and secretary, and has entire charge of the management of the journal and the direction of its policies. The Board appoints the Editor, selects the publisher, designates who shall act as business manager, etc. The Associate Editors are appointed by three constituent organizations,—to represent El Paso, New Mexico and Arizona, respectively. In 1921 the present editor was appointed, a business manager and publisher selected, and since that time no changes have been made in these.

Some confusion has arisen on account of the circumstance, entirely fortuitous, that the secretary of the Medical & Surgical Association of the Southwest and the editor of Southwestern Medicine, happen at present to be one person. This does not give the Medical & Surgical Association any rights not possessed by any one of the other organizations, with regard to the journal. The journal profits from this double relation, and the Association does not suffer, but either the Board of Managers or the Association can terminate this concentration of functions, any time they see fit, by placing some other person in one of these offices, or by placing two entirely different people from the present incumbent, in both of them.

ST. JOSEPH'S HOSPITAL, (Phoenix)

October Staff Meeting

The St. Joseph's Hospital Staff met on October 13, 1930, promptly at 8 p. m., with forty-five doctos present. The meeting was called to order by Dr. Brockway and the minutes of the previous staff meeting, and the executive council minutes, were read by Dr. Greer, secretary.

The problem of a meeting night was discussed. It was moved by Dr. Mills that we not approve the motion of the Maricopa County Medical Society, which had been carried previously, to change our meeting night from Monday to Tuesday night. Amendment was made to this motion, by Dr. Sweek, that it be necessary to bring the signature of fifteen members before the matter of changing the meeting night could be voted upon. This was included in Dr. Mills' motion by Dr. Mills' consent. The motion was seconded by Dr. Felch. After much discussion the motion was voted down, eight in favor and sixteen against. Dr. Jordan moved to retain Monday night as staff-meeting night and this motion was seconded by Dr. Bannister and upon vote there were thirteen for the motion and eleven against—many not voting. The chair ruled that the motion was carried and the meeting will continue Monday night as usual.

The following program was prepared by Dr. Frank Milloy:

Case No. 20387—Pre-eclamptic toxemia, presented by Dr. C. N. Ploussard, discussed by Dr. F. C. Jordon (See elsewhere in this issue.)

Case No. 18979—Fracture of right femur with separation of the lower epiphysis. To be presented by Dr. J. M. Greer, with open discussion following.

At the close of the discussion on Case No. 20387 there was no more time available and the meeting was turned over to the County Society.

J. M. GREER.
Secretary.

ST. JOSEPH'S HOSPITAL (Phoenix)

September Staff Meeting

The first meeting of the season of the St. Joseph's Hospital Staff was held September 15, with forty-one members present. Due to the illness of Dr. Brockway, chief of staff, Dr. Palmer presided over the meeting.

The regular monthly analysis of hospital service was read by Dr. Milloy.

The program for the meeting was prepared by Dr. Goodrich and consisted of the following:

Case No. 20296—Intestinal obstruction following appendectomy. The presentation by Dr. Vernon Kennedy, was followed by general discussion.

Case No. 20451—Post-traumatic tetanus; presented by Dr. T. W. Woodman, and discussed by Dr. Dr. R. B. Raney. (See elsewhere in this issue.)

Case No. 20262—Subphrenic abscess, presented by Dr. Frank Milloy, and discussed by Dr. R. J. Stroud. (See elsewhere in this issue)

J M. GREER,
Secretary.

BOOK REVIEWS

PHYSICAL DIAGNOSIS, by Richard C. Cabot, M. D.; Tenth Edition, 529 pages; price, $5; William Wood and Company, New York.

This important work is recognized as a standard of diagnostic methods and this 10th edition contains new material relating to coronary disease, electrocardiography, cancer of the lung, cardiac asthma, toxic hepatitis, and encephalitis lethargica. Revision has also been made of the chapters covering laboratory methods and these sections have been brought up to date. This is a most practical book in that it presents only those methods which have been found of definite value.

THE MEDICAL CLINICS OF NORTH AMERICA. May, 1930. Vol. 13, No. 6, W. B. Saunders & Co., Philadelphia and London. This number is contributed by the Mayo Clinic. The cases cover a wide range of subjects and the case histories are concisely yet thoroughly presented. There are many illustrations including reproductions of x-ray films, photomicrographs and numerous charts and diagrams. This number contains the volume index.

H. P.M.

ARTERIAL HYPERTENSION. By Edward J. Stieglitz, M.S., M.D., Assistant Clinical Professor of Medicine, Rush Medical College, University of Chicago, attending internist Chicago Lying-in-Hospital, assistant attending physician, Presbyterian Hospital. Cloth, price $5.50, 280 pp. Paul B. Hoeber, Inc., New York.

The material of this monograph is derived from a series of lectures given at the Rush Medical College for several years past. In presenting this subject, the author places special emphasis on the etiology and upon the physiologic pathogenesis, showing that hypertension represents a physiologic reaction occurring in disease of the smaller arterioles, arteriolar hypertonia being the fundamental change and increase in intravascular pressure is secondary. There are 21 illustrations and the bibliography and the bibliographic subject-index is very complete, so that the reader may readily refer to original articles for further study. H.P.M.

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(Continued from page 598.)

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ENURESIS
Review of Current Literature

(Continued from page 595.)

causes which operate to cause the internal ring to stand open.

The treatment consists in lifting the bladder floor and providing a tonic muscular sling to holld it up. This can be done very simply and easily by doing a perineorraphy in the male or female.

P. J. Crozier Griffith and A. Graeme Mitchell[14] write: "Most of the cases are a persistence of the condition normal to infancy which period is characterized by a powerful detrusor apparatus and an insufficient sphincter control of the bladder. A small number develop it after the normal control had been acquired.'" They describe the physiology of urination as a reflex act in which are concerned the bladder musculature, the efferent and afferent nerves, and the spinal centers for the nerves. Reflex stimuli may arise from pathologic conditions in any other part of the body and affect the detrusor muscle so as to call for urination. Finally there is the part played by the brain which should control urination even during sleep. Local irritations, inflammation, infestations, calculi, anatomical and urinary abnormalities, etc., may reflexly stimulate the detrusor muscles. Nervous excitability, general debility, malnutrition, anemia, glandular insufficiencies, over eating, etc., may also need to be seriously considered as important factors in the etiology of enuresis. Fatigue, over eating, sleeping on the back, too heavy covers, etc., may contribute to loss of the inhibitory conrol of the bladder during sleep.

Langley Porter and Wm. E. Carter[15] say: "Enuresis in older children when there is no definite condition that produces irritation of the genito-urinary tract, is essentially an expression of a maintained involuntary as opposed to a voluntary control. It means either that the child has never had the cerebral mastery of the pelvic viscera properly established, or that through bad environment and imperfect training, a neurotic habit has been encouraged; this happens even after cerebral control has been attained, with a reversion to the infantile involuntary type of bladder evacuation without central inhibition."

Lewis H. Adler[16], says that the etiology of enuresis may be one or more of a variety of conditions among which may be an incomplete development of the sphincter muscles, a debility of the neck of the bladder, abnormalities of penis or clitoris, cystitis, nephritis, pyelitis, vesical or renal calculi, inadequate control from the spinal cord nerve centers, glycosuria, rectal irritation, constipation, hyperacidity of the urine, over distention of the bladder, masturbation, genital trauma, and bladder relaxation which may be a local manifestation of general lack of tone of the entire muscular system from marked nutritional diseases.

McKee[17] says that the metabolic activity of the sensitive nerve cells is probably much disturbed and that diseases of the nerve centers are recognized as factors inducing enuresis.

Rachford[18] writes that in children enuresis is probably a true neurosis with a disturbance of the nerve centers in control of the bladder.

Nicholetopoulos[19] says that enuresis results from fatigue which causes children to sleep so heavily

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that they do not arouse when the stimulation from the full bladder occurs.

Leonard Williams[20] thinks that frequently a hypoactive thyroid is the cause of enuresis in both adults and children.

Ozerezkowski[21] has noted anesthesia of the bladder in a large portion of the cases.

Wachenstein[22] says that enuresis is probably essentially a habit development somewhat akin to tics and other spasm habits; it was frequently found associated with stuttering. He says that certain cases persist into adult life more often in females, and become hard to cure, as is the case with habit tics.

Monro[23] is of the opinion that heredity is a potent predisposing factor. In a family of six children the disorder seemed to be inherited from the father. He also says that two of the female children were more affected during the menstrual period; two others were subject to epilepsy.

Thiemich[24] says that enuresis is generally due to a nervous condition and that as a rule it is the children of neurotic parents who are affected with enuresis. He has observed epidemics of enuresis in institutions in which many children are grouped together.

Fuchs[25] has found occult spina bifida in 68.2 per cent of adults and 35 per cent of children with enuresis.

Constantiesco[26] observed two cases of nocturnal enuresis associated with renal tuberculosis and two other cases after nephrectomy.

Robson[27] had an incontinent woman age 20 in whom there seemed to be practically no bladder capacity.

Beilby[28] reports that three years observation in a state industrial school has made him form the conclusion that 90 per cent of enuresis is due to masturbation. He had 75 cases of enuresis in a total school population of 900.

C. E. McGork[29] says that in females it is important to examine the clitoris and perhaps to do circumcision. In nine cases he treated four women who had borne children. He has found a definite family tendency for enuresis.

J. Allen[30] divided the etiologic features into five groups; genito-urinary, nervousness, malformations, general and idiopathic. The urine may be at fault, either too acid or too alkaline; there may be pressure on the bladder or inflammation of various parts of the urinary tract; or there may be calculi, new growths, balanitis, vulvo-vaginitis, worms, and other local conditions. There may be malformation of the spinal cord or genitourinary tract. Among general causes he mentions rheumatism, thyroid insufficiency, diabetes, etc. He says that Still found enuresis associated with rheumatism in about five per cent of his cases.

Koplik[31] thinks enuresis is a functional neurosis; he has seen it persist into adulthood and present serious problems for treatment.

Holt[32] regards enuresis as a neurosis and thinks the solution is early training.

Editor[33] stresses the importance of excluding nocturnal epilepsy as the cause of nocturnal enuresis. If no organic basis is found the establishment of proper habit must be the solution of the trouble.

Isaac A. Abt, Herman M. Adler and Phyllis Bartelme[34] report in a study entitled "The Relationship Between the Onset of Speech and Intelligence" that "children who are enuretic at the age of three years or above learn to talk later than those who have established dry habits prior to that time."

(Continued in next Issue.)

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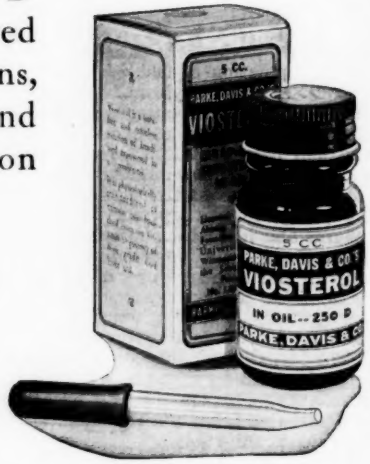

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